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LIVER STONES

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"There is no disease of the gallbladder ducts. The only disease is that of the liver cells themselves."—Chauffard. 1890.

SEEING extensive liver changes in only mild cases of infection so astounded this talented clinician as to cause him to make the above statement. Even 40 years ago when gallbladder surgery was still in its infancy, experienced clinicians, because of apparent facts, concluded that gall duct disease, unless considered as associated with the liver parenchyma, was difficult to understand, evaluate and to establish prophylactic treatment.

Although the problem of hepatogenesis has made great progress toward solution during the last 40 years, it has not been satisfactorily, or finally, solved as yet. Genetic fundamentals in liver stone disease are of great importance because liver stones are not only found while operating upon the bile ducts or parts of the liver itself, but are also found at autopsy in people who have never been operated upon at all or who have died of some other disease.

Notwithstanding that isolated cases have been described in the literature of the sixteenth and seventeenth centuries, there is not as yet a completed statistical résumé of this condition, and surgeons with the most extensive material of gallbladder surgery at hand have reported only a few isolated cases. Even Beer's (1904) very extensive work cites only one or two case histories from the literature. Frerich, in 1861, presented five cases and von Thudicum (1863) and von Niemer (1891) published five cases of liver abscess in which stones were found. These are the only cases we have knowledge of which have been presented during the last century. Beer describes six instances occurring in his own series which, however, were found only at autopsy. Peters, Huhendorf and others have reported cases of liver stones which were also reported by Courvoisier, who collected 50 cases in 1891. We purposely do not cite reports from the latest foreign authorities in this paper. They are numerically and alphabetically arranged in an appended reference list.

Körte's monograph in 1905 and in 1928 presents only a few isolated cases

which are included in our appended statistical list. All the reports from the Russian literature date back into the twentieth century and deal with the description of a few isolated cases by Romanzew, Grasmik, Mattrossow, Calusow and Terebinskij, while the balance of the cases are of a more general character, their authors describing certain definite and specific phases of gallbladder and duct disease. These reports are by Walynzew, Hesse, Kadjan, and Fedorow (case taken from the work of Prof. Ikonnikow and Jukelson). My own work on this subject more fully completes these statistics by adding four more cases taken from the Clinic of Director Prof. Martynow, where I was an assistant, and another case which was placed at my disposal by Prof. S. P. Fedorow to be used in this work. These cases can be found in the "Brief Communications" from the service in the Annals of the Surgical Hospital and also in the Kliniceskaja Medicina, November 23 to 24, 1930 (Russia).

I was prompted to try to collect the following case reports as completely as I could, in order to ascertain the possibility of a preoperative diagnosis in hepatolithiasis; which type of applied method of treatment would be the most definitely satisfactory for the best end-result; the pathologico-anatomic picture; and finally, to clear up a number of questions of the pathogenicity of gallstone disease as a whole.

Our own cases consist of five in number. Forty-one are taken from the foreign literature and 16 cases are Russian. This report, I think, represents the most complete collection of cases, both foreign and Russian, on this subject. The only cases which will be detailed are the five of our own. The others will be referred to by number and the author's name only (references).

These figures do not by any means exhaust the existing material on the subject. Courvoisier's report, however, was not considered in our study because some of the cases were doubtful and not clearly described, their description resembling more the usual picture of gallbladder perforation with entrance of stone into the liver tissue, rather than that of liver stones. In other cases, the authors erroneously accepted hepatic duct stones as true liver stones. Furthermore, the surgery of the bile tracts in Courvoisier's report was not correctly designated by the authors, as no stones were found in the liver and no stones had ever actually been present. Although these authors may have been right, they concluded that the extrahepatic stones they did find originated in the liver.

Walijew critically evaluated gallbladder surgery in his work entitled "Liver Stones," the material for the treatise being taken from the Clinic of Professor Bogoras and reported at the First Congress on Surgery in North Caucasia, Rostow-Dow, 1926 (Russia).

Bykenhelm and Digby reported eight cases of liver stones found among the Chinese. Only three cases, however, were essentially liver stones, while the other five can only be accepted as of probable intrahepatic origin without any conclusive substantiatory evidence.

I have not detailed the cases reported by Erdmann, 1914. The report consisted of a critical review of 270 operations on the biliary tract with one

case of recurrence of stone after a cholecystectomy, which he classified as a true recurrence of stone from the intrahepatic ducts and cellular tissue. Cases of this kind are frequently seen. Kehr's statement that he has never seen a true recurrence of stone of intrahepatic origin may not be entirely correct but it is decidedly wrong to ascribe "overlooked," or "forgotten" stones as a true recurrence.

Italian and Spanish literature of the last year is not included in our statistics because the original articles were not at hand and the respective information came to us only from the yearly reports of cases (Finochietti, Lang, Hartinam, Weber and others). I also included a number of other cases, which, although not specifically described as liver stones, can, by their course and manifestations, be placed in this category (Eickhoff, Büdinger and others).

Individual authors (Calusow) dispute the rare occurrence of hepatolithiasis without conclusive grounds and principally only on the basis of logical and mathematical proof. Hepatolithiasis, according to data from Pathologic Institutes, is of rare occurrence; nevertheless, it can be accepted that a more careful examination of the liver in a number of these cases may have proved the exception.

We take the following from extensive statistics: Courvoisier, 1903. 16,025 autopsies—Found 1,714 cases of cholelithiasis in which there occurred one case of liver stone. Wolynzew, 1904. 945 autopsies—12 cases of cholelithiasis—two cases of liver stones. Hesse, 1911. 17,402 autopsies—278 cases of cholelithiasis and one case of liver stones. Miyake, 1913. 8,406 autopsies—257 cases of cholelithiasis—20 cases of liver stones.

In Japan, liver stones are relatively frequent. Miyake collected his material from three Pathologico-Anatomic laboratories. He found 191 cases of cholelithiasis in Tokyo, with 13 cases of liver stone; 35 cases in Kioto, with one, and 31 cases in Kinschu, with six instances of liver stones.

The veterinary literature reports only a small percentage of hepatolithiasis and cholelithiasis. Simonesco, 1901, found cholelithiasis seven times in 260 human autopsies; and in 3,000 autopsies on oxen he found cholelithiasis twice.

Case histories in the veterinarian field show the striking rarity of this process in animals, and particularly in domesticated animals. Wild animals, such as deer and hares, rarely have cholelithiasis. It appears the anthropoid apes never have cholelithiasis. In the case of Dedjulin, a piece of wire, 3 cm. long, was found in the gallbladder. Sommermeyer states that he found dilated intrahepatic ducts filled with stones and intestinal contents in two cases of cholelithiasis in horses. These stones were essentially made up of bilirubin and calcium and contained cholesterol in lesser amounts. Frequently, foreign bodies such as corn, sand, fine nails, wire, *etc.*, have been found in the gallbladder.

In our 57 collected cases, 25 were operated upon as well as autopsied; 16 cases were interval operations without autopsy; and 16 cases came to autopsy without operation. In a number of instances, stones were only found incidentally at autopsy. A case reported by Hesse is in this category,

where the patient died of peritonitis following an operation for gastric carcinoma; also a case by Beer, where the patient died from sepsis; and finally, our own case (Case IV) where death followed a rib resection for empyema. The autopsy in this case showed stones in the liver and bile ducts. The last two cases of Fabre and Petrisso, 1932, did not come to our notice until after this work was ready for the press. The results in these two cases do not, however, alter our conclusions.

Pathologic Anatomy.—The pathologic anatomy is most marked by essential changes in the liver. Schematically, these liver lesions can be divided into the following groups or stages:

Group 1.—Isolated lesions in portions of the liver with firmly fixed stones.

Group 2.—Circumscribed foci in the liver, with stones imbedded in a cavity containing bile, pus, or a (greasy) pultaceous mass of thickened bile.

Group 3.—Presents a more extensive process and the liver stones are coexistent with extrahepatic duct involvement, while the intrahepatic ducts contain a pultaceous (greasy) substance with occasional occurrence of stones of the same material, or of different structure and consistency than that of the stones in the extrahepatic ducts.

Group 4.—The same picture as in Group 3, except that the small biliary channels present the character of a purulent cholangeitis and in some cases the liver will be studded with foci of small abscesses containing stones either fully formed, or of a cement-like, or pasty, consistency.

The third group, in some cases, as in the fourth, is quite frequently accompanied by marked and severe changes in the cardiovascular system, the kidneys and the liver, leading to complete cessation of function of these organs. In some instances, it appears as though one of the groups was a prodromal stage to the next group, with manifestations of extensive pathologic changes; gradual, quantitative and qualitative increase of the process involving the greater part of liver parenchyma and liver cells and showing evidence of early sedimentation, for later stone formation, in the most minute intrahepatic ducts by a definite inflammatory process. The liver appears normal. This macroscopic appearance, however, frequently differs from the palpatory as well as the microscopic findings (Fig. 1).

The marked, existing pancreatitis sometimes, when the liver appears normal at the operation, concentrates one's entire attention on the pancreas. At autopsy, however, immediately following the operation, deep seated and long standing changes in the entire liver parenchyma, with the coexistence of many well formed stones, will be found. At times the liver will be found normal at autopsy in the presence of marked cholangeitis and also a large number of stones in the bile ducts where the presence of a normal liver should naturally be excluded. External appearance of the liver does not exclude liver pathology. Microscopic examination should be done in every case to clear all doubt (Figs. 2 and 3). In the majority of cases the liver is enlarged. Its color is dark blue (Calusow), with indurated edges at times or definite anemic foci (Büdinger), icteric (Haberer), markedly hyperemic

FIG. 1.—Macroscopically changed liver tissue (somewhat enlarged), occurring in the first case we operated on. This portion of the liver was removed, containing stones in chain, or tandem formation. There is a clear picture of proliferation of Glisson's capsule and signs of liver jaundice due to stasis.

(Kehr, Büdinger and others) and, more rarely, its appearance is soft and flabby (Rufanov) or sometimes it appears in the form of a constricted liver, corset liver (Oehler).

Enlargement of the liver is twofold in character; either a general uniform enlargement, or irregular enlargement of only one lobe, as seen in our first case where the liver was not of conventional form and the right lobe extended to and into the pelvis, the left lobe presenting an unusual incisure and a small accessory lobe measuring 3 by 3 cm., which was suspended from its under surface; this half of the liver was particularly subjected to pathologic changes. Oehler also speaks of such increase in the size of the liver, especially of its inferior surface. This enlargement is sometimes caused by a preexisting cavity filled with pus, bile and stones. It is usually either a cyst, a pus pocket or a carcinomatously indurated area and with any one of these three diagnoses the patient is usually operated upon. The indurated nodules in the liver may reach the size of a hen's egg (Naguchi), or the liver may have spots of whitish placques or scar tissue which, upon incision, display either a stone or a cavity. These cavities at times besides containing stones may also contain pus and bile, or either alone (most frequently altered bile) and the stones are then found imbedded in the wall of the cavity. At times the tensely dilated ducts, filled with stones, branch out from the liver cavity.

The wall of the cavity is rarely thin or easy to open like a cyst or an abscess depending upon its relation to the surface of the liver and its location. Most frequently, its wall is firm and compact and removal of all the stones is only fully accomplished by resecting portions of the liver parenchyma itself. It is sometimes formed by dilated intrahepatic ducts, or, in some cases, may consist of scar tissue only.

Macroscopic liver changes, in the form of obliterated, regenerated areas, may, in some cases, be recognized as soon as the liver is incised. Liver changes usually coexistent with cholecystitis are microscopically present only when the case is marked and advanced. One will find conditions of liver stasis, hyperemia, biliary cirrhosis, foci of round cell infiltration, sometimes abscesses (recognized microscopically only), scars, atrophy of the parenchymatous cells, necrosis and regenerated foci of newly formed tissue. The cells themselves contain a branch-like distribution of pigment. Intrahepatic ducts are dilated in most cases, containing pigment and sometimes a proliferation of liver connective tissue itself around the bile ducts or, in other words, the picture of pericholangeitis is found and seen (Eickhoff). Sometimes the smallest and most minute branches of the bile ducts are filled with a doughy mass, or filled with very small, yet formed calculi, presenting the picture of a lithiac cholangeitis.

The pathologico-anatomic picture of the liver is the result of complications arising from recurring or perforating cholecystitis with changes in its surrounding tissue, or is at times found where there has been an extension of the process to neighboring and adjacent organs and tissues. Hydrops of the gallbladder was never seen.

Very marked changes in the gallbladder wall were sometimes coexistent with the absence of gallstones in the gallbladder itself. The microscopic picture of the gallbladder also corresponds to the same changes one finds in cholelithiasis.

Bile.—There is usually only a small amount of turbid bile found in the gallbladder because of either the presence of a stone, cholangeitis, or an atrophic and shrunken gallbladder (Büdinger, Rufanov and others), or the bile is of a mucoid (Kadjan), mucopurulent (Beer and others) or purulent character (Beer and others).

Cases of perforation of the gallbladder into the liver have been observed. The bile, not infrequently, contains clumps of bile pigment or cholesterol and sometimes its content is made up of a glycerinated (Bykenelow), clay colored, lardaceous mass, in which bile pigment is held suspended (Körte) yet sometimes this mass is grayish-white or green and cholesterin-like in color (Büdinger). Sometimes, shortly after operation there occurs only a slight amount of bile drainage and bile secretion which shortly disappears completely. In some cases only white bile was found. The quantity of bile in the bile ducts was mostly found to be small, but in a few cases we found that the liver capillaries contained a great deal of bile; on the whole, and most frequently, however, there appeared to be a deficient liver function and a lowered bile secretion.

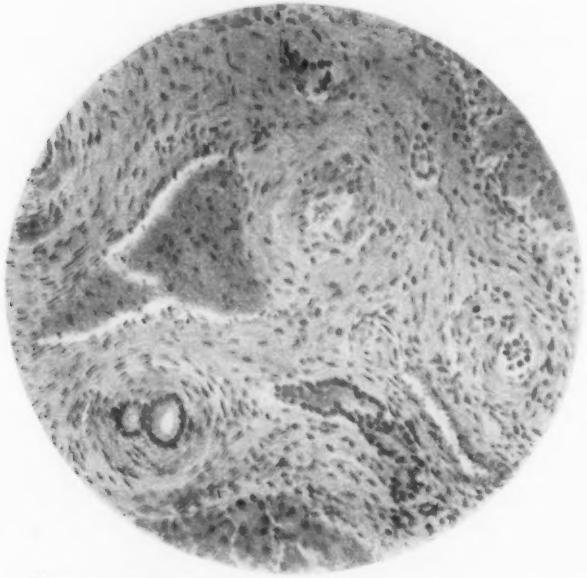
The bacteria found in the bile belong to the same group found in cholelithiasis, such as *B. coli* (Vachel and others), coli-like bacilli (Naguchi), spores (Matrossow), *Staphylococcus* (Matrossow), *B. pyocyaneous* (Kadjan), *Streptococcus viridans* (Eickhoff). The bile was not examined bacteriologically in all cases.

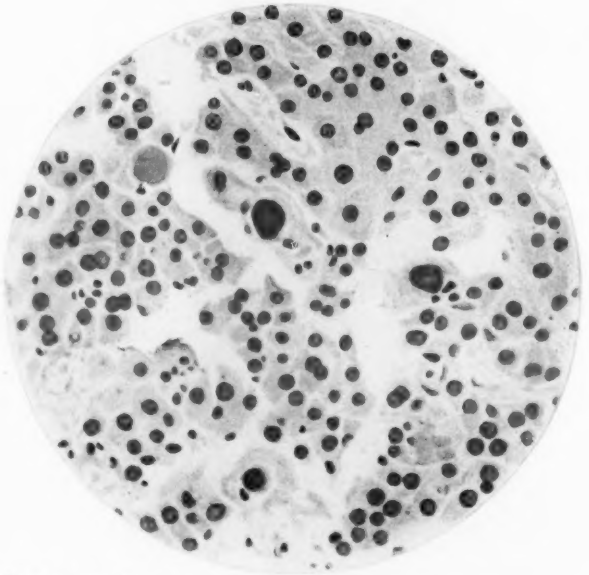
Neighboring organs and tissue are frequently involved in the process in the form of adhesions ranging from mild perivisceral to firm and formed adherent scar convolutions. The predominating adhesions involve the stomach and subsequent adhesive scars may sometimes lead to permanent pyloric stenosis demanding gastro-enterostomy. In two cases a gastric ulcer was found at autopsy.

Intestinal adhesions sometimes form an adherent mass analogous to that seen in circumscribed purulent peritonitis, from which the clinical manifestations of a complete intestinal obstruction occur, indicating the necessity of performing an enterostomy.

Obstruction in the liver sometimes causes sufficient kinking of the intrahepatic ducts to predispose to bile stasis and the formation of stone above this obstruction (Teribinskij). Edema or scar tissue of the hepaticoduodenal ligament has been observed (Fedorow), the scar formation favoring disturbance in the portal vein circulation and stasis, hemorrhage, or ascites (Kehr and Eickhoff). Sometimes the smallest bile ducts and passages are found dilated, forming rather large cavities with a glossy inner surface containing pigment, mucus, stones and white bile.

Cases of neoplasm in the bile ducts have been seen. Granular, brownish-





FIGS. 2 and 3.—The biliary capillaries contain compact stones which have a somewhat dark center and a lighter periphery. In some of the biliary capillaries, there is evidence of stasis. Atypical liver structure; lobuli not definitely circumscribed. In parts of the liver, the central veins are absent and there is a characteristic, atypical, cirrhotic picture. In the liver cells themselves, one sees dilated canals.

black, pigmentary deposits are mostly met with in the presence of a parenchymatous, necrotic or necrobiotic area and are due to excretion of pigment from the liver cells. The intrahepatic ducts on the liver surface were sometimes dilated to an enormous size, in some instances to the diameter of a goosequill, and immediately upon incision of the liver there ensued a flow of bile. As we mentioned before, the stone-containing bile ducts present the appearance of a firm and fixed chain of stones.

Bile Ducts and Gallbladder.—Marked anatomic changes are present, mostly involving the extrahepatic ducts. Macroscopically, they are rarely found normal. These ducts, as well as the intrahepatic ducts, are mostly dilated to large circumferential dimensions. The cystic duct may contain valves and diverticula (Oehler) in which stones of such large size (in Oehler's case, the size of a pigeon's egg) may be found, so imbedded that they can be removed only with great difficulty. The cystic duct may also be tensely filled with a compact mass of stones, or, as in the case of Kadjan and others, stones may be present in the liver and the cystic duct found completely occluded, indurated, distended, or in some cases, pulled over to the stomach by contracting scar tissue. The common and hepatic ducts, due to obstructive tissue changes, may develop stasis on the one hand and duct dilatation on the other; the sequence of infection and the presence of stones.

Dilatation of the bile passages reaches large dimensions, even to the size of the circumference of the small intestine (Jukelson). The walls of the biliary passages, due to their distention, *etc.*, as mentioned above, lose their elasticity, become thickened, infiltrated and sometimes (Körte) ulcerated or encrusted. Less frequently we find scar tissue changes, such as stricture of the common and hepatic ducts. In a number of cases the gallbladder remains normal or sometimes it is found flaccid or distended. In the majority of cases, however, marked anatomic changes are present. In 40 per cent of cases, evidence of acute, purulent or ulcerating cholecystitis or an empyema of the gallbladder is present; 20 per cent show either an atrophied, shrunken, or indurated wall and sclerosis of the gallbladder and finally, in 20 per cent, the gallbladder will be cicatrized and occluded, the occlusion sometimes being confined only to its mucous membrane as in the case of Eickhoff, or involving its entire wall. Pericholecystitis is, not infrequently, observed.

In secondary involvement of other organs we find, first, changes in the pancreas in the form of a chronic, secondary, indurative pancreatitis (swelling of the head). The percentage of chronic pancreatitis in this condition seems to be less, however, than we usually find in cholelithiasis. Acute pancreatitis was not present in any of our cases. Secondly, there may be a right-sided pleural involvement in the form of a serous or purulent exudate (pleuritis), as a complication during the course of this process.

Finally, the kidney may be damaged (Rufanov). The urine may contain albumin, leukocytes, erythrocytes and kidney epithelium. Cases of kidney cirrhosis with nephrosis and nephritis with edema and anuria, as well as uremia, have been described. We did not find any instances of acute peri-

tonitis in any form. Eickoff found a chronic progressive peritonitis in his case. In a number of instances the alvine inflammatory reaction confined itself to a localized, purulent peritonitis.

Stones.—The number, character, size and shape, as well as the chemical structure of the stones occur in a great, diversified multiplicity. The occurrence of stones in the liver and in the various portions of the extrahepatic passages is shown in Table I.

TABLE I

STONES COEXISTENT IN LIVER AND EXTRAHEPATIC DUCTS

	Gallbladder	Cystic Duct	Hepatic Duct	In Liver	Cases
1.	Gallbladder	Cystic duct	Yes	In liver	19
2.	Gallbladder	Cystic duct	None found	In liver	5
3.	Gallbladder	None found	None found	In liver	2
4.	Gallbladder	None found	None found	In liver	4
5.	None found	None found	None found	In liver	6
6.	None found	None found	None found	In liver	6
7.	None found	None found	None found	In liver	11
				Total	53

It will be seen from this table that in 20 per cent of cases there were stones in the liver and none in the gallbladder and bile passages. In 80 per cent of cases there were stones in the gallbladder or biliary passages, or in both at the same time, as well as in the liver. In 35 per cent of cases there were stones in the gallbladder, bile passages and liver at the same time, and in 60 per cent the gallbladder had stones, whereas in 40 per cent there were none present. These figures, in the study of the clinical course of the process and operative result, are of great importance, as will be seen later.

The number of stones varied from one to 1,000 or more. The presence of minute and very small stones caused thought that their numerical assessment was impossible, or that the liver was completely filled with them. Removal of a number of stones at operation did not prove that all the stones had been removed, because frequently at a second operation more stones were found and removed, or they were expelled after the operation, through the drains. There are two cases recorded, where 12, 20, 36 and 52 stones were found.

In our third case, upon incising the liver, stones projected from all the smaller and larger biliary ducts. In the case of Vachel and M. Stewens, there were 520 stones found in the deformed part of the liver, one of which, weighing 270 Gm., lay in a cavity with other small stones. In this case, the hepatic and common duct contained up to 5,100 stones, of which the largest weighed 100 Gm. The total weight of all the stones was 1,378 Gm.

In most cases, however, we find small stones with gravel, ranging in size from cherrystone or pea size, and measuring from a few millimeters to 1 cm. in diameter, in the form of a pultaceous-like, sebaceous mass. In some cases, the stones are as large as a hen's egg and of two of these stones removed from a liver scar one was 2.7 cm. and the other was 3.2 cm. in diameter.

In a number of cases (Federow and others), all intrahepatic passages, as

well as the extrahepatic bile ducts and biliary passages, were tensely distended by a pultaceous, unguentous mass.

The color of the stones varies according to the amount of their pigment content, from a grayish-white (Hawks; Matrossow), yellowish-white (Büdinger); yellow (Rufanov); yellowish-green (Büdinger); gray (Matrossow); dark red (Beer, Rufanov, Kocher); brown (Büdinger); brownish-black (Rufanov) and black (Beer, Calusow). At times we find light, as well as dark stones, in the same case.

The shape of the stones is most frequently round, sometimes club-shaped (Körte); dome-formed, ovoid (Beer); multicornered, (Sieber); polyhedral (Rufanov); tetrahedral; and they may be only partly (Beer) or entirely facetted (Sendler, Romanzew). Furthermore, cylindroid stones have been found, as well as arborescent forms, representing the smallest bile passages (Terebinskij, Barth and others).

The stones may be soft and easily pulverized, yet sometimes, as in the case of Büdinger, the soft, powder-like stones began hardening 24 hours after removal, and at the end of three to four days had taken on the character of firm calculi.

The Chemical Consistency of the Stones.—Stones occur in various combinations of their main constituents as bilirubin or cholesterol, or they occur as pure cholesterol or bilirubin stones. Cholesterol stones were found in the cases of Sieber, Büdinger, Lewison (48 cholesterol stones), Judd, Burden and others. Pigment and pigmented cholesterol stones (Beer and others) are also met with. In our case, there was a calcium combination with bile pigment, bilirubin and cholesterol, with calcium and magnesium salts. Terebinskij found the same combination, with the exception of magnesium, in his case. Beer's case of extrahepatic stones contained an encrusted pigment mass around a cholesterol center. The intrahepatic stones, on the other hand, were devoid of the cholesterol center, being made up of pigment and calcium.

In Kadjan's case, all the ten stones contained 68 to 76 per cent cholesterol and 17 to 24 per cent calcinated bilirubin. One stone in the common duct contained a center with a high cholesterol content. In Matrossow's case, the single, round, gray stone consisted of a carbonate phosphate of cholesterol and pigment. In Büdinger's case, the stones were made up of cholesterol and a small amount of calcium and bilirubin. The large stone in Vachel's case, which weighed 270 Gm. and which was one of 250 other stones, contained 18 per cent cholesterol, 39 per cent calcinated pigment, and 0.01 per cent iron.

In Oehler's case there were two kinds of stones: (a) small, facetted, cholesterol stones containing bile pigment, and (b) large firm stones of which two were 4 by 2 cm. and 5 by 2 cm. in size. The large stone contained a nucleus of cholesterol and pigmented calcium. Two others, however, contained no center. Romanzew found 30 facetted stones, in size from gravel to

a cherry-pit, with large, pigmented centers, surrounded by a radiating cholesterol layer.

ETIOLOGY AND PATHOGENESIS: The question of cause and formal genesis of hepatolithiasis and cholelithiasis are the same. It revolves around hypothetical reasoning and practical interest. The primary cause, whether arising from partial (gallbladder), or complete (liver); from local or general conditions of the organism; from stasis; from infection; diathesis or disturbed organic metabolism, has not as yet been solved.

Does stasis arise from anatomic conditions or anomalies (Berg); or is its occurrence due to functional disturbances (Westphal)? If the clinical history discloses infection, is this the immediate cause of the inflammatory process in the bile passages and the subsequent liver tissue destruction? Is the liver damage, primary or secondary to a metabolic disturbance (hypercholesteremia) which produces toxic changes and disturbs the normal quantitative and qualitative secretion of bile?

Mindful of the fact that every third person after 70 is a gallstone carrier, the question is raised of whether arteriosclerosis affects the vessel wall and brings about changes in the contracting force of the excretory ducts and disturbs the nutrition of the liver cells thereby acting as a primary cause. On the other hand, is the arteriosclerosis and subsequent stone formation only the result of a disturbed cholesterol or metabolic dysfunction?

Is the frequency of lithiasis in the female due to tight lacing; corsets, or other mechanical means of pressure, or is it due to an unstable and predilectively susceptible nervous system increased by the rising and falling curve of cholesteremia which concomitantly changes with and from the beginning of puberty; during every menstruation and every pregnancy during the sex life of every woman? How does the vegetative nervous system act on liver metabolism? Is it solely through secondary, stimulative, sensory, motor and secretory nerve cell and fiber disturbances which convey abnormal impulses not only to the functioning liver apparatus and ducts but also to other organs? Or conversely, can lithiasis be ascribed to lesions and stimulation in the central or the local nervous system (solar plexus) causing colloidal changes, changes in mineral metabolism and in the permeability of the cell membrane itself, thereby establishing a primary cause for stone development?

These are the essential controversial points we find in the present literature, around which the genesis of lithiasis is centered. As has been previously stated, stones are frequently found in people of advanced years.

Literature frequently refers to the presence of gallstones as of theoretical interest only. When infection or occlusion of the excretory ducts occur, however, they assume practical importance because of the serious and sometimes threatening clinical picture of cholelithiasis. If, on the other hand, in cases of latent cholelithiasis, treatment is instituted before the inflammatory process has developed, this practical interest cannot be considered.

When active and definite formation of calculi begins is not known, and

it is impossible to establish or ascertain when the initial clinical symptoms first began. Not infrequently the clinician accepts a functional liver syndrome or one coming from the bile ducts, *i.e.,* the sphincter of Oddi, or accompanying manifestations coming from other organs, stomach, intestines, pancreas, etc., with or without organic disease, as lithiasis. Actually, however, we frequently cannot determine the syndromic termination of the physiologic pathology and the clinical onset of the pathologic anatomy. A preexisting mild infection, an occlusion of the ducts, or an additional infection may be sufficient to kindle or stir up a definite clinical picture after the previous course of a portentous process. The very fact that a stone is present demonstrates marked metabolic disturbance which may be still further activated by the stone itself.

The presence of stone can be accepted as a stage only, and not a separate group, of manifestations in gallstone disease. The clinician should not allow himself to become interested. It represents the same stage in the clinical exhibition of gallstone disease as that of the stage of grease and gravel as the forerunner to the formed gallstones.

It is not my intention to summarize the theories on formation of stone. They have been fully elucidated by Lütkens, Fedorow, Finkelstein and Bereskin. The latter has critically evaluated the theories and observations of the foreign and Russian literature, Aschoff, Naunyn, Chauffard, Rovsing; their predecessors and schools have by a series of intense studies of factors substantiated one or another of these theories. The theories of infection, stasis and disturbed metabolism, as of principal etiologic importance, constantly vary for and against their evaluation and acceptance. Each theory is of preeminently primary or essential etiologic moment, and is upheld by a series of valuable clinical and experimental observations. Each of these conditions may occur in certain people, probably in each patient at a certain age, from a congenital, acquired, anatomic or functional anomaly of the liver and biliary ducts and a subsequent stasis.

Infection and disturbed metabolism may be present, however, without the actual formation of stones. Sometimes an attack of lithiasis occurs without any apparent etiologic factor. In these cases, conditions which predispose to stasis and infection should be looked for in the anamnesis.

For 50 years, disturbed metabolism was considered the main etiologic factor in lithiasis. Today this theory has been replaced by accepting stasis and infection, the latter playing the leading rôle, as the main cause in this condition. Fedorow and others defend Chauffard's theory. It is hard to conceive or visualize the entire course and process of stone formation beginning and active only in the bile ducts and gallbladder, to the considerative exclusion of the liver, the vegetative nervous system; the metabolism and all conditions which influence the quantitative and qualitative characteristics of the bile before it reaches the gallbladder, and finally not to forget to include the various degrees of colloidal change and permeability of the liver cells.

We divide our material of hepatolithiasis into two groups: (1) those complicated with cholecystitis and cholelithiasis; (2) those without any complication of the gallbladder. We found a differing clinical course and a different result of the surgical technic in one and the other group. Complicated extrahepatic duct lithiasis with hepatolithiasis considered by its course, as well as the result of its therapeutic and surgical treatment, is a more severe illness than that of hepatolithiasis occurring alone.

Leopold presents a case of liver stones in the absence of a gallbladder, thus establishing the possibility of the formation of stones in the liver when the gallbladder is absent. In a case of removal of a large stone from the common duct, in the congenital absence of a gallbladder, Finkelstein reported the recurrence of a stone the size of a pigeon egg which he removed. This stone, without doubt, came from the liver. In Terebinskij's case, the center of a gallbladder stone was made up of products of duct effusion. These facts led us to conclude that liver stones possibly do form without any gallbladder pathology.

In our third group of cases of liver stones, the extrahepatic ducts were completely intact. Stasis in this type of case can, therefore, play no part in etiology and infection can hardly be looked upon as a primary cause. If the infection were virulent or purulent, cholangeitis would follow, and a mild infection would not only involve the extrahepatic ducts, but the liver as well. More likely, the primary condition in these cases is due to disturbed metabolism.

Other concomitant conditions may exist, however, but they cannot be classed as a primary cause. In this conclusion, I omit any consideration of hunger, fever, *etc.*, relative to their influence to bile secretion, stasis or the qualitative character of the bile (Experiments of Wilishanin, Michailow, Lukjanow). Russian authors have shown that hunger, fever, uremia and kidney lesions decrease the secretion of bile. Lukjanow presents the observations of Uffelmann, in the case of a woman with a cholecystenteric fistula, who showed marked cessation of bile secretion during an intercurrent attack of pneumonia.

It is a physiologic fact that pain itself will reflexly check liver activity. Siebert and Castaing (1899) state that gallbladder colic acts reflexly on the liver, causing cessation of its function (analogous to an anuria during kidney stone colic) with the appearance of sugar and urobiligen in the urine. In these cases, we can also observe qualitative changes of the bile and deposits of its solids due to changes in the alkaline medium, besides the condition of stasis. It is known that acid bile precipitates bilirubin (Thudicum). Infection, as well as a number of other conditions (metals, *etc.*) can alter the reaction of the bile. The findings of Ikonnikow and others have substantiated pigmentary deposition outside of the gallbladder. Every damage to liver parenchyma, as well as change in its vascular supply, influences the permeability of the liver cells. The complicated innervatory relation to the various sections of the extrahepatic ducts should be recognized as particu-

larly important because of the active duct contraction it induces. Westphal has confirmed the clinical significance of these dyskinesias as a cause of biliary colic and resultant lithiasis.

Excitation of change in the regulatory bile secretion apparatus during menstruation is the example of endocrine influence. Molinar, basing his opinion on observation in 365 of his own cases, believes that constant or temporary cessation of genital gland activity is one of the most frequent causes of gallstone disease.

Cases of lithiasis with a psychoneurotic background are well known, and conversely, there is no doubt that the secretory function of the liver is greatly influenced by psychic conditions as is manifested in cases of emotional jaundice.

Stuber remarks that the stimulation may arise from humeral influence (hormone or toxines) or may originate from some portion of the autonomous nervous system, the vegetative or the highest cortical centers. The result of this excitation or stimulation of the organ itself, especially in women with a high lability of their vegetative nervous system, is essentially manifested where there is the greatest amount of nerve supply, namely, the sphincter of Oddi and Lutken. Stasis, obviously, will follow motor disturbance.

Where heretofore possible stasis followed by the deposition of concrements due to changes in physical, chemical and colloidal characteristics of the bile was accepted as a conjectural probability, today this question appears to be definitely solved.

Change in the innervation of the liver can lead to stone formation. In this respect, the experiments of Doctor Gaisinsky in the laboratory of Professor Alperin are of great interest. He concluded that visceral innervating disturbance of the liver essentially through the sympathetic alters the 24 hour cholesterol balance in the organ notwithstanding what the blood content is. Due to this action of the sympathetic, the cholesterol permeability in the liver is changed, owing to disturbance of cholesterol metabolism, or probably due to changes in the permeability of the liver cells themselves.

Conditions which produce this variation in the 24 hour cholesterol balance bespeak alteration for liver permeability without, however, disturbing the periodic 24 hour quantity of the bile and cholesterol content from its normal level. By modification of innervating influences to the liver, changes in the permeability of its membrane for colloidal cholesterol were produced by abnormal influence of the sympathetic on the hepatic artery and complete section of the hepatoduodenal ligament. Gaisinsky observed the formation of pigment and mineral deposition in the bile: agreeing with Rovsing as to the genesis of lithiasis. These pigment deposits can predispose to stone formation. Alperin by his experiments emphasizes the importance of the sympathetic nervous system in changing the physicochemical character of the bile. Liek lays great stress on the nervous system. He points to a number of motor and secretory disturbances such as spasm, hypertonia,

copious cholesterol and mucous secretion, delayed absorption, similar to the type seen in membranous colitis or in bronchial asthma. Gastric sub- or anacidity are in close relation to a neurogenic influence in three-fourths of the patients whose gastric peristalsis was studied during gallstone attacks.

Stasis in the liver may be caused and the origin of liver and gallbladder stone may arise following change in liver function, change in the reaction of the bile itself, change in the colloidal portion of the excretion, as well as alteration in the permeable qualities of the liver cell membrane.

Rovsing's theory of the cause of liver stones, is confirmed by a large number of cases. The contention of Minkler, Tietze, Körte, Finkelstein, Rufanov and others, that although the parenchyma of the liver exhibits changes in the presence of extrahepatic duct infection and cholelithiasis, the liver pathology is not, as we will see later, caused by an ascending infection from the gallbladder to the bile and smallest ducts, and thus into the liver, but is more likely to occur from primary changes in the liver itself, producing conditions favorable to stasis and subsequent lithiasis. Disturbance in metabolism primarily brings about a change of function in the liver and not in the bile ducts which confirms this contention. This, however, does not exclude the possibility of an ascending infection, an angiocholitis or a liver abscess, the onset of which may be hastened by stasis or decreased secretion of the liver cells.

The evalution of our material offers interesting conclusions. Thirty-three per cent died of cholangeitis (7 in 21); 66 per cent of liver insufficiency (14 in 21); liver damage in its relation to bile duct pathology was in the majority. We failed to find any basis for the statement that under all conditions stone formation begins only in the gallbladder. The same conditions and the same factors may exist in the smallest, as well as the largest, bile ducts (Hein, 1846, as cited from Romanzew). To say that the primary predisposing factor for stone formation is the presence of a cavity where stasis or infection may more easily occur or enter is analogous to excluding the parenchyma of the kidney as the most important site in the formation of kidney stones. Many cases of pyelitis exist for years without the formation of calculi. The same contention holds good in the stagnant urine in the presence of a large prostate where even, with a large bladder cavity, stasis and infection do not cause formation of stone. It is also a pertinent question in cases of hydrops of the gallbladder. In these cases, the history is sometimes negative relative to a report of any attacks of gallbladder colic; in some cases only a dull pain or a very slight attack of colic has occurred, although the stone has advanced into the neck of the gallbladder, causing the hydrops by obstruction.

It seems that with a normal gallbladder wall with active contractibility, any closure of the cystic duct by a stone should manifest more clinically pronounced symptoms than are usually observed. The wall of the gallbladder is frequently only slightly atrophic, from the distention of the

hydrops, yet, however, there is a concomitant coexisting marked liver change. Whereas we accept the usual explanation of mechanical closure of the cystic duct, we can hardly refute the contention of another cause, such as the entrance of a stone into the gallbladder, which originated in the liver, gradually increasing in size in the neck of the gallbladder, with a consequent gradual distention of this viscus which retains relatively good contractibility of its walls. Kulenkampf (1926) is probably right when he says that it is impossible for a stone to travel from the gallbladder into the ducts considering the structure of Heisters' folds.

In 1930, I stated that the climatic and living conditions of various peoples in their relation to the marked difference in the frequency of incidence of liver cirrhosis and gallstones, especially in the formation of variously chemically constituted stones, such as pigment and cholesterol, should not be ignored. At the last Pathologico-Anatomic Congress, Askanasi, discussing liver cirrhosis in relation to the macro- and microlithic interest, called attention to the contentions previously put forth by Hoppe Seiler (1866), Klebs (1869) and others, of the presence of copper in the pigment stones. The large amount of copper found in the livers of people living in Geneva caused the author to reopen this question of the cause of this condition. Askanasi, furthermore, brings up the question as to whether this metal does not play the rôle of a catalyzer in the oxidation process, which function, according to Naunyn, accompanies infection.

Askanasi, in relation to macro- and microliths and the liver cirrhosis, shows that in 21 cases of liver cirrhosis he found microliths 15 times and pigment stones three times. Lammel, however, in 1927, found microliths 21 times in 62 cases of cirrhosis and pigment stones nine times. In searching for the cause of the numerous instances of cirrhosis occurring in Geneva and Japan, the author contends that this is possibly explained (as compared to other countries) by the large copper and iron deposits in these countries. Mallory, in 1925, has experimentally substantiated the clinical observations of the author.

Referring to the reciprocal relationship between the liver and spleen, we also see a parallel between liver changes and stone formation, although it is a different pathologic consideration. This relationship is not without importance. Moynihan, in one of his cases quoted by Finkelstein, after six operations on the gallbladder and gallducts, during which time he removed several thousand stones, drained the ducts and, finally, removed the spleen before he obtained a cure. Another similar case is reported by Bernhard and Malet-Guy (Klarel, Lyon, Surgery, 1928). A gallbladder fistula was produced because of pigment stones in the gallbladder. The formation of the pigment stones was coexistent with a hemolytic icterus. Due to the continued icterus, the spleen was removed following which the patient rapidly recovered.

This confirms acceptance that stones in the gallbladder and ducts may sometimes arise from causes exogenous to the gallbladder and the liver,

as substantiated by cited cases; the cells of the latter being unable to take care of, or react to, the increased demands made upon them, the spleen reacting to the increased breaking up of hemoglobin and iron deposition with consequent formation of true pigment stones.

All these facts concerning the possible method of the intrahepatic origin of stones, the liver changes in extrahepatic gallstones, microliths and their relation to metabolism and the rôle of the spleen and its functional products toward the formation of liver stones, belong to the consideration of the genesis and the etiology of gallstones. It is not to be forgotten, however, that the liver can also react to a large number of damaging factors, *i.e.*, endocrine in character (Molnar, 1926), disturbance of its metabolism, diseases of the blood, and other influences which in one way or another cause stone formation by connective tissue proliferation.

(*To be continued*)

CONGENITALLY SHORTENED ESOPHAGUS (THORACIC STOMACH)

Report of Two Cases Found Postmortem

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A number of malformations of the diaphragm associated with abnormalities of the esophagus and malpositions of the stomach has been reported in the literature; but only in a very limited number of such cases has it been possible to examine the organs postmortem and determine the exact relations of the parts. The cases here presented offered such an opportunity; and, because of the clinical importance of any facts which might lead to a better understanding of the origin and significance of these disturbances, it is felt that the following detailed description of the anatomic structures is justified.

Two cases were found during 1934 and 1935 among the dissecting room specimens in the University of Texas Anatomical Laboratory.

Case Reports

Case I.—The subject was that of a Mexican female, F. A., aged 60 years. The death certificate gave bronchopneumonia as the cause of death; beyond this fact no clinical history was obtainable.

No external indication of any abnormality was apparent except a fair degree of scoliosis, convex to the right in the lower thoracic region with an associated compensatory curve in the lumbar region. The body was short, stocky, and quite obese—the latter condition indicating that the alimentary tract had functioned satisfactorily.

From the thoracic aspect, it was noted that there had been displacement of a heart of normal size to the right. Measurements showed that it extended 5.5 cm. to the right of the midsternal line at the level of the fourth costal cartilage and only 4 cm. to the left at the level of the fifth interspace.

When the lungs were removed, a soft tumor mass beneath the mediastinal pleura could be seen in the posterior mediastinum. The tumor measured 7 cm. by 10 cm., and the wall of the sac over this mass was wrinkled and appeared as if it had been distended even to larger dimensions during life (Fig. 1).

When the sac surrounding the mass was incised vertically along its midventral line, it was seen to be lined with a glistening serous membrane which proved to be continuous with the peritoneum below. The contents, consisting of the stomach and the major portions of the greater and lesser omenta, were not fixed at any point to the wall of the sac, except near the middorsal and midventral lines where the serous membrane lining the wall of the sac reflected on to the stomach (Fig. 2). This reflection near the midventral line was a part of the lesser omentum. A finger was placed against the left side of the stomach and carried downward through a constriction at the diaphragm into the great peritoneal sac.

An incision was then made through the lesser omentum, and the cavity to the right of and behind the lesser omentum was explored. A finger was passed dorsally and met another reflection from the wall to the contents. By placing a finger on either side of this dorsal reflection, it was possible to observe that the two layers were adjacent, short, and held the stomach closely against the dorsal wall, except for a short distance along the body

of the stomach. This lower portion of the body of the stomach had apparently undergone partial rotation on a vertical axis. It was only in this limited extent that abnormal greater and lesser curvatures could be identified. Below the body of the stomach, the pyloric portion was bound closely to the dorsal body wall. A finger was then carried downward behind the lesser omentum along the right side of the stomach and pushed through the constriction of the diaphragm into the cavity of the omental bursa. Hence, it was apparent that both the great peritoneal sac and the omental bursa were carried upward surrounding the high placed stomach. Above, at about the level of the lower end of the esophagus, the serous membrane which lined the sac was reflected on to the contents.

FIG. 1. (Case I.)—Shows tumor mass in the posterior mediastinum.

FIG. 2. (Case I.)—Shows part of contents of sac. Note the dorsal reflection from the wall of the sac on to the stomach.

Dissection of the wall of the sac showed that it consisted of two layers of tissue (Fig. 3). The outer layer was fibrous in character. It was adherent all around with the subjacent layer; but a definite separation could be made easily. This outer layer thinned out as it ascended to the esophagus and spent itself by forming a delicate fibrous sleeve about the lower end of this organ. Inferiorly, the layer became thicker, contained occasional muscle fibers, and lower down became continuous with the tissue of the diaphragm. The second layer or lining was a serous membrane, the peritoneum; and the reflections and connections of this layer in the sac have already been pointed out.

Further dissection revealed an esophagus flattened from before backward and wider than normal, the width varying from 2.5 cm. to 3 cm. There was no evidence of stricture. The esophagus took a straight course vertically downward. After coursing a distance of about 1 cm. within the sac, the esophagus ended at the level of the third costal cartilage by terminating in the high placed stomach. With reference to the vertebral column, the esophagus ended at the level of the lower border of the body of the sixth thoracic vertebra. That the esophagogastric junction was at this level was verified by microscopic examination of the mucous membrane.

The vagus nerves united on the esophagus to form a definite plexus (Fig. 3). The

plexus began at the level of the upper extent of the lung roots and extended downward to the point at which the esophagus entered the sac. The right continuation of the plexus placed it posteriorly on the esophagus; and some branches, after penetrating the wall of the sac, reached the stomach along the right side and passed ventrally on the organ (Fig. 3). However, most of the branches continued inferiorly between the two layers of the wall of the sac, passed below the main part of the diaphragm, and terminated in the celiac plexus. The left continuation of the plexus passed downward without branching for 2.5 cm. superficial to the sac on its lateral aspect. Branches were then given off which took one of two directions: a few branches took a recurrent course upward toward the esophagus and then turned downward along the left side of the stomach; but most of

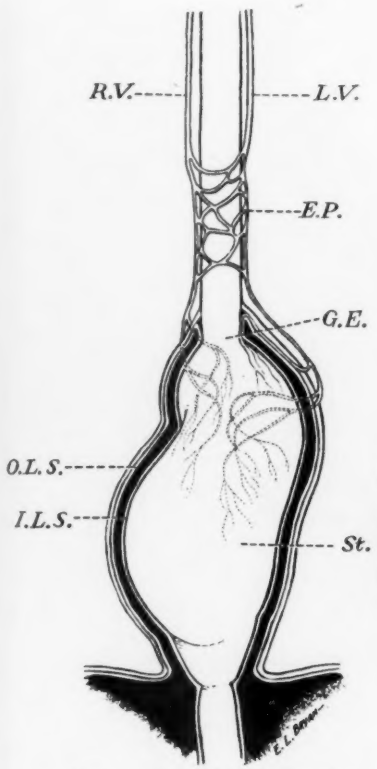

FIG. 3. (Case I.)—Diagram showing wall of sac and arrangement of vagus nerves. *R.V.*, right vagus; *L.V.*, left vagus; *E.P.*, esophageal plexus; *G.E.*, esophagogastric junction; *St.*, stomach; *O.L.S.*, outer layer sac; *I.L.S.*, inner layer sac.

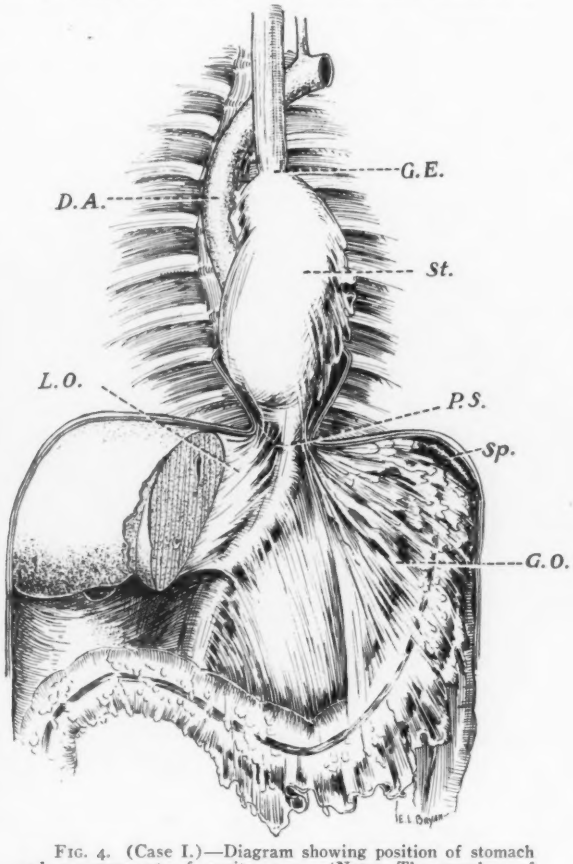

FIG. 4. (Case I.)—Diagram showing position of stomach and arrangement of peritoneum. (Note: The number of thoracic vertebrae and ribs shown is not accurate.) *D.A.*, dorsal aorta; *G.E.*, esophagogastric junction; *St.*, stomach; *P.S.*, pyloric sphincter; *L.O.*, lesser omentum; *Sp.*, spleen; *G.O.*, greater omentum.

the branches turned posteriorly, going deeper and deeper into the wall of the sac until the serous membrane was reached. The nerve fibers followed this serous layer dorsally until it became reflected on to the stomach; here these fibers were distributed to its left side.

The pleural and pericardial sacs showed no significant abnormalities. When the abdomen was examined, the rudimentary greater omentum was found knotted up in the left hypochondriac region. The liver was normal in gross appearance and location.

Only a very limited portion of the stomach showed from the abdominal aspect (Fig. 4). The pars pylorica extended vertically downward to the sphincter which was located 1.5 cm.

below the constriction at the diaphragm and 2 cm. to the right of the midline of the body. The duodenum continued downward for a short distance and curved slightly to the right to become situated along the right side of the vertebral bodies. This position was kept until the body of the fifth lumbar vertebra was reached where the gut crossed the midline to the left and ascended to the left side of the body of the second lumbar vertebra to the level of the duodenojejunal flexure. The gut below this flexure was normal in position.

The inlet to the sac-like dilatation of the diaphragm was oval shaped (2.5 cm. by 3.5 cm.), the long axis of the oval being placed transversely in front of the vertebral column at the level of the lower border of the tenth thoracic vertebra. Ventrally, the margin of the inlet was fibrous in character, while the dorsal boundary was muscular—the muscle fibers intervening between the inlet ventrally and the descending aorta and the vertebral column dorsally. The portion of the pars pylorica within the inlet was about 2.5 cm. in diameter, the remainder of the constriction being snugly filled with parts of the lesser and greater omenta.

The peripheral attachments of the diaphragm were normal. No abnormality other than the sac-like dilatation about the high placed stomach was noted.

The peritoneum below the transverse mesocolon presented no unusual features; but in the upper abdomen the relations and connections were peculiar. They can best be appreciated by reference to Fig. 4. The omental bursa possessed significant limitations.

Above the transverse mesocolon, the peritoneum was reflected upward as a single layer covering the dorsal body wall. Near the midline this layer ascended over the duodenum and pancreas and on upward through the constriction at the diaphragm over the pars pylorica. In other words, the pars pylorica and the duodenum were bound down by a single layer of peritoneum to the dorsal body wall; neither possessed any sign of a dorsal mesentery.

Along the right side of the upper part of the duodenum (for a distance of 7 cm. below the pyloric sphincter) and at a higher level along the ventral surface of the stomach, the peritoneum passed ventrally and slightly to the right as one layer of the lesser omentum. The layer became reflected upon itself to form the epiploic foramen (of Winslow) and to become continuous with the peritoneum lining the omental bursa. The so called "right free" border of the lesser omentum, together with its contents, had the appearance of being on the stretch and ran almost in the horizontal plane, thus maintaining in part the embryonic position (Figs. 4 and 5).

The omental bursa was found to be very limited. Inferiorly, it reached no farther than the level of the lower extent of the epiploic foramen which was about at its normal level. To the left the bursa was limited near the midline of the body where the posterior or right layer of the lesser omentum became reflected on to the duodenum and the pyloric portion of the stomach and then farther posteriorly on to the dorsal body wall. The omental bursa was found to continue upward through the inlet into the recess to the right of the stomach—as has already been pointed out.

Along the left side of the pars pylorica, beginning just above the sphincter, the sheet of peritoneum was brought forward and folded upon itself. This folding became quite redundant higher up on the stomach, being attached above to the rotated portion of the stomach along its greater curvature. The redundant double layer of peritoneum passed inferiorly through the constriction at the diaphragm and was connected below to the left half of the transverse colon as far to the left as the left flexure. Above the flexure the peritoneum was reflected about a spleen normal in size and position. After passing around the spleen and flexure, the left layer reflected laterally on the under surface of the diaphragm, left kidney, and body wall, while the more medial layer turned downward and medially and became continuous with the sheet ascending from the root of the transverse mesocolon.

The gastric circulation is worthy of note. The arterial supply along the left side of the stomach came mainly from the right gastro-epiploic. This vessel ran upward more or

FIG. 5. (Case I.)—Note the absence of the stomach and the horizontal direction of the "right free" border of the lesser omentum.

FIG. 6. (Case II.)—Shows tumor mass in posterior mediastinum.

less parallel with but about 3 cm. to the left of the stomach, giving branches over to the organ—mainly to the pars pylorica; a few branches passed downward toward the transverse colon and distributed themselves in the rudimentary greater omentum. No left gastro-epiploic artery could be identified. The splenic artery, however, gave off several small gastric branches; these coursed upward retroperitoneally through the constriction at the diaphragm to reach the upper part of the dorsal aspect of the left side of the stomach. Veins accompanying the above arteries were small.

On the right side of the stomach the main blood supply was from the left gastric artery, which arose normally from the celiac artery which coursed upward and ventrally. The left gastric artery came into the sac posteriorly near the midline, between the outer fibrous and inner serous layers, and extended upward as high as the esophagus, to which a small branch was given. The main vessel turned downward on the stomach between the layers of the lesser omentum. The right gastric artery was normal in origin and very small.

The coronary and right gastric veins were unusually large, perhaps as a result of partial suppression of venous return by pressure from the boundaries of the inlet to the sac during times of distension. Both of these veins emptied into the portal.

The pancreas was at its usual level and was essentially normal in position.

Case II.—This subject was another Mexican female, F. E. No clinical history was obtainable except that she died at 70 years of age from cardiorenal disease.

The body was short, stocky, and obese. There was slight scoliosis, convex to the right in the upper thoracic region with a compensatory curve to the left in the lower thoracic and upper lumbar regions, combined with a mild lordosis in the lower lumbar region.

A soft tumor measuring 8 cm. by 10 cm. and extending as high as the lower border of the body of the fifth thoracic vertebra (opposite the sternal end of the third interspace) showed in the posterior mediastinum. The heart, normal in size, was displaced to the right.

The sac about the tumor contained better than half of the stomach and considerable parts of the greater and lesser omenta. The peritoneal connections within the sac were strikingly similar to those found in Case I. A narrow mesentery held the stomach close to the dorsal wall of the sac. This dorsal mesentery became continuous below with the greater omentum which was attached along the greater curvature of approximately the lower one-half or rotated portion of the stomach. The lesser omentum extended from an attachment near the ventral border of the stomach to the ventral wall of the sac, the line of attachment to the wall of the sac running to the right as it extended inferiorly. Below the level of the diaphragm the lesser omentum passed to the right and stretched from the porta hepatis to the lesser curvature of the rotated portion of the stomach.

The short esophagus became continuous with the stomach at the upper extremity of the sac. The esophagus was wider than normal, measuring 3 cm.; the width was uniform except below, where the esophagus was constricted to 2 cm. as it joined the stomach.

The inlet to the sac-like dilatation of the diaphragm measured 7 cm. transversely by 4.5 cm. anteroposteriorly and lay opposite the body of the tenth thoracic vertebra.

The liver, which appeared to be twisted to the right and displaced in that direction, was smaller than normal, particularly the left lobe. The pancreas and spleen were normal in size and position. In reaching the stomach, the arterial and nervous supply followed essentially the same course as found in Case I.

The great peritoneal sac was normal in significant features except for the extension of the space upward into the diaphragmatic recess anterior and to the left of the stomach. Likewise, the omental bursa narrowed and continued upward to the right of the upper part of the stomach retained in the recess.

Summary.—(1) In each case the esophagus was shortened, ending caudally, in Case I at the level of the sixth thoracic vertebra and in Case II

FIG. 7. (Case II.)—Shows part of contents of sac.

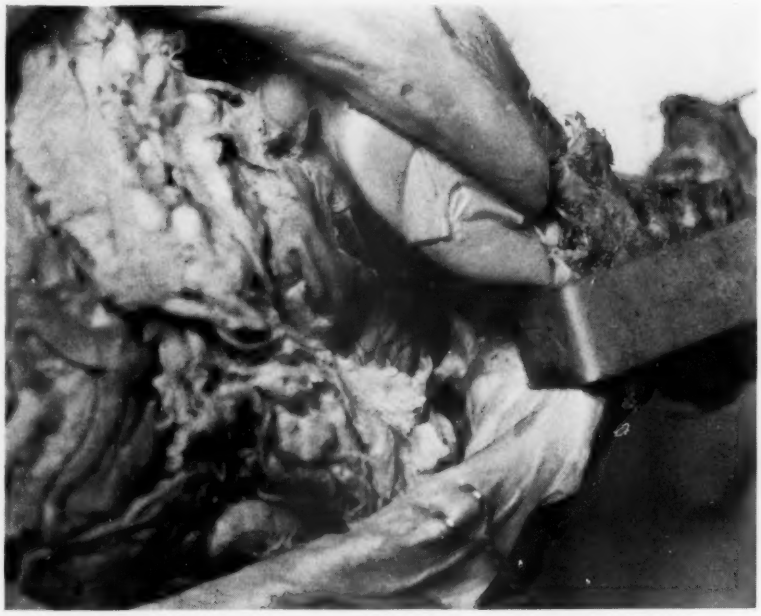

FIG. 8. (Case II.)—Only about one-half of stomach is visible from abdominal aspect. Note inlet to sac.

ending at the level of the fifth thoracic vertebra; and a large part of each stomach was fixed above the usual level of the diaphragm.

(2) In both the diaphragm was essentially normal except for the sac-like dilatation stretched over the high placed stomach.

(3) The upper portion of each stomach was largely in an unrotated, vertical position—as evidenced not only by the position of the organ but also by the connections of the peritoneum, by the course and distribution of the vagus nerves, and by the arrangement of the gastric blood vessels.

(4) The greater and lesser peritoneal sacs were continued upward into the diaphragmatic recesses about the high placed stomachs.

Discussion.—It is not possible to determine with certainty the mechanism of production of such anomalies as here seen; but, in view of the manner of development of the body cavities, foregut, and diaphragm, and in view of the arrangement of the parts in these cases, I believe that these abnormalities at the so called esophageal orifice are the result of a developmental arrest which took place during early embryonic life. While I have no new thought to offer concerning the probable primary cause of these developmental upsets, I feel it will be helpful to review briefly pertinent, diverse opinions in this regard and to point out the varied terminology used in describing such cases.

Primary Causal Factor.—In 1919, Bailey[1] reported a case in which he found, during the dissection of a man 77 years of age, the stomach in the thorax. Bailey explained the positions and relations of the parts "by supposing that the anlage of the stomach lay abnormally far anterior on the alimentary canal." Such an explanation can neither be proved nor disproved.

Since the pleuroperitoneal canals of the embryo connect the pleural and peritoneal spaces, persistence of part of either of these canals has been used to explain the cause of certain so called herniae. An improper closure of one of the pleuroperitoneal canals more likely gives rise to an absence of diaphragmatic tissue between the costal and vertebral attachments of the diaphragm with direct communication between the pleural and peritoneal spaces and with the esophagus going through the esophageal opening normally.

Fraser[2] states that Browman, according to a private communication, feels that the infracardiac bursa may form at least a part of such a "para-oesophageal recess" and further suggests that the "muscle round the oesophageal foramen is either imperfectly developed, or has retrogressed (rückgebildet) and that abdominal pressure then forces up a pouch."

Other observers have considered improper development of the esophagus to be the primary cause, and I feel that this explanation is most likely. Papers from this point of view have been written by Clerf and Manges,[3] Monkhouse and Montgomery,[4] Findlay and Kelly,[5] and others.

In the cases here reported, suppose that, for some obscure reason, the esophagus in development failed to reach its normal length; and perhaps the esophagus widened to an abnormal degree—as we see in these specimens, the excess width possibly being related to the shortening. Subsequently, when the elements of the diaphragm were displaced caudally from the primitive

cervical level, instead of the esophagus elongating sufficiently to permit the stomach to be carried to its usual level, the stomach was anchored at a level higher than normal; and the elements of the diaphragm became stretched over the stomach to form a sac-like pouch. Since the peritoneal portion of the original coelom is about the primitive stomach, we should expect to find (as we did in these cases) the sac-like pouch of the diaphragm which stretched over the stomach to cover also this serous membrane about the stomach.

The unrotated position of the upper portion of each of these stomachs, associated with rotation to the left of parts of the lower portion, seems to indicate that the descending elements of the diaphragm caught the upper part and contributed toward inhibition of normal rotation; but the lower portion, which was reached later by the descending elements of the diaphragm, was allowed to rotate.

It is interesting to note that the portion of the dorsal mesogastrium which contained the spleen underwent a shift to the left in both cases. In Case I this shift with rotation of the adjacent part of the stomach occurred in spite of lack of rotation above or below. Hence, it appears that the migration of the spleen is to a large extent independent of the rotation of the stomach and is perhaps important in influencing the shift to the left of the dorsal mesogastrium to begin the formation of the greater omentum.

The question may be asked: Is the scoliosis exhibited by each of these cases related to the gut and diaphragmatic anomalies? I simply raise the question.

Terminology.—Complexity in development of the involved parts has led naturally to the use of a varied terminology in describing such abnormalities as here seen.

As already indicated, improper development of the esophagus has been regarded as the fundamental or primary cause. Hence, we see these cases referred to as the "congenitally shortened esophagus" or "short esophagus." These terms enjoy wide usage.

Perhaps the largest number of these cases has been diagnosed and recorded as "non-traumatic diaphragmatic herniae." However, the term "hernia" is now being reserved by some writers to apply to those cases in which the stomach or other abdominal parts were once down at their normal level and subsequently displaced to a higher level.

In a recent article Jackson and Jackson[6] referred to a like condition as "supradiaphragmatic stomach." In their discussion they also use "hiatal hernia of the stomach" with apparently the same meaning.

A very similar condition, if not identical in essential features, is found in the case reported by Fraser.[2] He terms the abnormality "para-oesophageal recess of the diaphragm." Fraser thinks his case is the same type as one recorded by Gruber[7] for which he proposed the name "recessus retromediastinalis phrenicoperitonealis." Fraser and Gruber have offered terms which are more accurate anatomically; but they are long and not in general use.

In the case reported by Bailey[1] he employed the term "thoracic stomach."

So far as I have been able to determine from the literature, this is the first recorded instance of the possibility of the stomach developing at a thoracic level. The term "thoracic stomach" has become quite generally used in referring to similar cases. LeWald[8] wrote an excellent paper on thoracic stomach from the standpoint of the roentgenologist. More recently, Goodall and Hoyt[9] reported five cases and gave an admirable discussion of the clinical manifestations of this condition. In addition to these observers, Lyons,[10] Morris,[11] and others have reported cases and employed the term "thoracic stomach."

"Thoracic stomach" has the advantage over other suggested terms in that it is brief and can be remembered as a developmental disturbance in which the stomach has been retained at a level higher than normal. It must be admitted, however, that the term is not altogether satisfactory. Strictly speaking, it is not accurate. There is no continuity between the pleural or pericardial and peritoneal cavities. The stomach in such cases is not in the thoracic cavity but within an extension of the abdominal cavity. The stomach is below the overstretched diaphragm. On the other hand, the stomach lies above the normal extent of the abdominal cavity and is at a thoracic level.

No doubt additional terms other than those mentioned have been employed. Suffice it to say, a varied terminology is being used to describe very similar conditions.

Frequency.—This variation in terminology used in reporting such cases makes it almost impossible to obtain accurate information concerning their frequency. The two cases reported here were found among 75 cadavers. Examination of the esophageal orifices and the esophagogastric junctions in all of these cadavers showed a third but less marked case. While I do not mean to infer that one out of every 25 cadavers will show some shortening of the esophagus with retention of part of the stomach at a level higher than normal, I do believe that such abnormalities are more frequent than is now generally recognized; and I trust that the report of these cases will serve to stimulate anatomists, pathologists, and clinicians to make more careful observations of the organs involved.

The author wishes to express his appreciation to Professor H. O. Knight for permission to publish these cases and to Professors Knight, Donald Duncan, and John G. Sinclair for their helpful suggestions and criticisms.

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EXTENSION OF GASTRIC CARCINOMA INTO THE DUODENUM

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THE majority of text-books of surgery and surgical pathology describe, very often quite dramatically, the abruptness with which the extension of gastric carcinoma stops at the pylorus. For example, Babcock[1] writes, "The growth tends to spread upward from the pylorus along the lesser curvature . . ., but it has no tendency to invade the duodenum." DaCosta[2] states, "Even in cancer of the pylorus the duodenum is seldom invaded." Thomson and Miles[3] write, "The growth is arrested abruptly at the pyloric ring and shows no tendency to invade the duodenum." Romanis and Mitchiner[4] write, "Here (the pylorus) the disease shows no tendency to spread into the duodenum." Boyd[5] states, ". . . the spread stops short only when it reaches the duodenum." Many other authors could be cited, but these few will suffice to emphasize the idea that is generally accepted.

Verbrugghen[6] has recently reviewed the material at the Mayo Clinic and called attention to the extension of gastric carcinoma toward the cardia along the submucosa beyond the visible edge of the tumor. He advised that in resecting the tumor at least four cm. of normal gastric wall should be allowed from the edge of the ulcer along the lesser curvature toward the cardia. In very diffuse carcinoma there may not be any visible edge and a proper point for resection could not be indicated. He says nothing about duodenal extension.

Since many surgeons believe that only a very small margin of duodenum need be resected for carcinoma in the distal third of the stomach, and since so many text-books state that the duodenum is immune from extension of gastric carcinoma, I have reviewed some of the microscopic preparations from both surgical and autopsy material collected during the past 30 years. Before analyzing this material, however, a review of the literature including the reasons for the prevalent point of view may be in order.

Rokitansky[7] in 1861 laid down the law, as it were, when he said that pyloric cancer was exactly bounded by the pyloric ring, and never reached beyond that to the duodenum. Both Kocher[8] and Mikulicz[9] believed very strongly in the integrity of the duodenum and stated that the duodenum could be resected with safety five to ten Mm. from the pyloric valve.

In 1865 Brinton[10] was the first to take exception to this rule. In 125 cases of pyloric cancer he found ten that had invaded the duodenum for a variable distance, often as much as an inch or two. He also cited a case of Lebert, making 11 cases in all. Carle and Fantino[11] found the duodenum invaded for distances of one to three cm. in three out of 14 cases. Cuneo[12] studied eight gastrectomies and found one in which the duodenum was grossly invaded; in

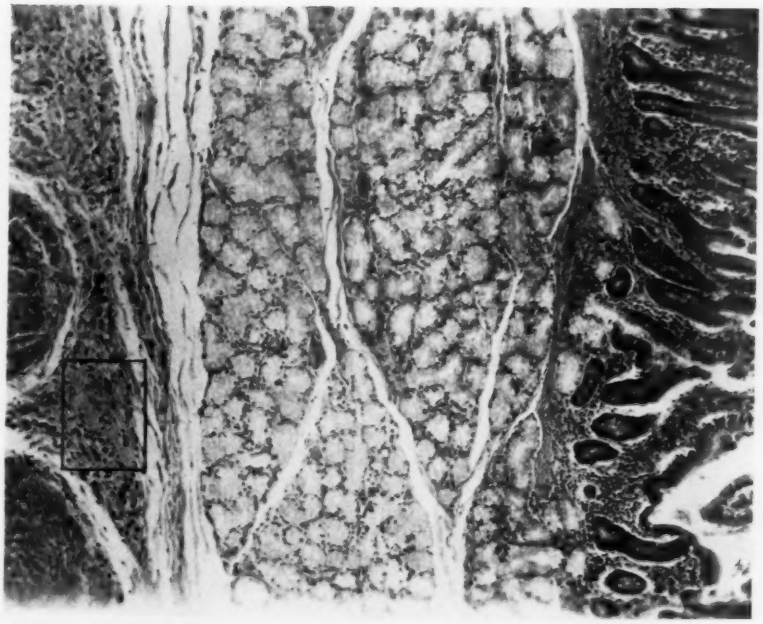

FIG. 1. (Case 14.)—A low power photomicrograph of a section of duodenum showing two large collections of carcinoma cells between Brunner's glands and the muscularis.

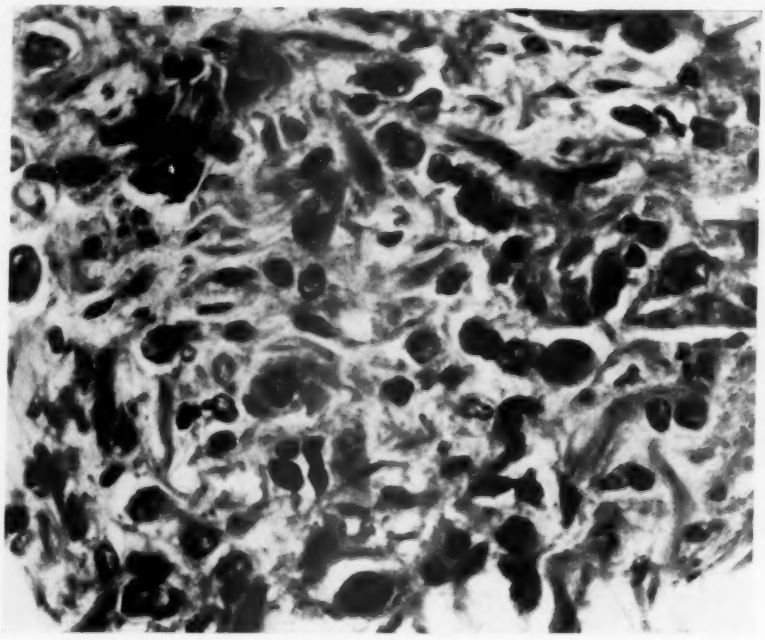

FIG. 2. (Case 14.)—A high power photomicrograph of the blocked area in Fig. 1.

FIG. 4. (Case 8.)—Photomicrograph of a section of the duodenal wall showing carcinoma within the lymphatics. Note the three small lymphatics in the upper left hand corner.

FIG. 3. (Case 10.)—Photomicrograph of a section of duodenum showing carcinoma diffusely infiltrating the muscularis. Note Brunner's glands in the upper left hand corner.

three of the remaining seven there was some invasion, the greatest being one cm. In 1901 Borrmann[13] studied 63 cases and found the duodenum invaded in 20, in nine of which involvement was at the line of resection, and in two, over two cm. from the pylorus. Moynihan[14] cites many of these references and emphasizes quite strongly the necessity of removing most of the first portion of the duodenum.

Statistical Data.—From 1900 to 1931 at the Massachusetts General Hospital there were 65 autopsied cases of carcinoma involving the pylorus. In many of these cases sections from the tumor only, without duodenum, were preserved. In reviewing these cases, however, it was possible to find six in which the growth had invaded the duodenum.

During the years 1919 to 1934 inclusive, the laboratory received 134 surgical specimens of stomach resected for pyloric carcinoma. In some specimens, as in the autopsy group, sections of the duodenum were not preserved, but making no allowance for these, 15 specimens of the 134, or 11 per cent, showed duodenal extension.

In 1934, when special attention was given to this subject, there were seven out of 28 surgical cases, or 25 per cent, that showed duodenal extension. This high percentage emphasizes quite clearly the necessity for the pathologist to take sections of the duodenum in all cases of prepyloric carcinoma.

The 21 cases thus selected, the six from the autopsy group and the 15 from the surgical group, all showed microscopically definite carcinoma for varying distances beneath Brunner's glands (Figs. 1 and 2). The extension rarely involved the mucosa, but spread along the submucosa (Fig. 3), often in lymphatics (Fig. 4). The extent of the duodenal invasion was measured in millimeters from the microscopic slide and because of the marked shrinkage in the tissues, due to fixation and dehydration, the figures were corrected to correspond to the gross specimens. This degree of shrinkage for tissues fixed in Zenker fluid, dehydrated in the alcohols and xylol, and imbedded in paraffin, was found to be approximately 20 per cent.

Nine out of the 16 pylorectomies showed tumor cells at the extreme edge of the resection, apparently indicating that there was still carcinoma present in the remaining duodenum; in only six was normal duodenum present beyond the farthest deposit of carcinoma. The sections from the autopsy cases were cut too short, because all six showed tumor at the extreme cut edge.

Discussion.—The significance of the statistics presented in the Table I is quite obvious; namely, that the surgeon should resect more duodenum. The first portion of the duodenum measures on the average five cm. This length should allow the surgeon to resect at least three cm. beyond the pylorus and still leave enough to fold in in the distal portion. If this procedure were followed there certainly would be fewer specimens showing tumor at the distal cut edge and less recurrence along the line of closure. In one case where the infiltration extended for 23 Mm. the surgeon was able to palpate the thickened duodenal wall, but usually the infiltration is so slight that only careful microscopic examination can detect its presence.

TABLE I

Case No.	Distance of Duodenal Extension (in Mm.)	Normal Duodenum beyond Carcinoma	Case No.	Distance of Duodenal Extension (in Mm.)	Normal Duodenum beyond Carcinoma
1*	4	0	12	6	0
2*	10	0	13	6	+
3*	12	0	14	6	+
4*	14	0	15	7	+
5*	17	0	16	8	0
6*	First portion	0	17	11	0
7	4	0	18	11	0
8	4	+	19	13	?0
9	5	0	20	13	0
10	5	+	21	23	0
11	6	+			

*Autopsy

SUMMARY AND CONCLUSIONS

(1) Twenty-one cases of carcinoma of the pyloric end of the stomach are presented in which there was extension of the tumor into the duodenum. Fifteen of the cases were partial gastrectomies, and six were autopsy cases.

(2) The degree of extension varied from four to 23 Mm. and in most of the surgical specimens carcinoma cells were found at the distal cut edge, implying that the remaining duodenal tissue contained tumor.

(3) It is suggested that at least three cm. of duodenum should be removed with all gastric resections for carcinoma at the pylorus, despite the fact that many of the text-books of surgery and surgical pathology state that gastric carcinoma does not extend beyond the pylorus.

(4) These findings indicate quite clearly the need for careful microscopic examination of the duodenum in all cases of pyloric carcinoma.

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GASTRIC AND DUODENAL PERFORATION DURING HOSPITAL TREATMENT

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Patients with known gastric or duodenal lesions perforating while in the hospital under standard, acceptedly efficient medical regimen are rarely encountered. Six such cases have occurred in Receiving Hospital during the last 15 years, and five of these within a recent three month period. The first was included in a previous report.[1] Another case of unsuspected ulcer perforated acutely while undergoing hospital treatment for an unrelated condition. Five of these were benign lesions and two were malignant. That the mortality rate should be 100 per cent is somewhat disconcerting, especially when, as Shawan[2] has shown, we have had unusual opportunity for experience in diagnosis and treatment of such cases. (His report included 227 acute perforations to January 1, 1933. Since that time this number has been increased to 289 as of July 1, 1935.) However, we believe it has been of distinct value to us to review these cases together as some unusual features become emphasized.

Case Reports

Case I.—B. S., a white male, aged 46 years, was admitted March 3, 1921, complaining of pain in the stomach, belching and heartburn. For eight years he had been having intermittent attacks of dyspepsia of four or five days' duration. During the previous year he had had almost constant moderate pain throughout the day, with severe pain coming on about three hours after the evening meal, accompanied by heartburn and relieved by vomiting. He avoided heavy and fat foods. There had been no hematemesis or jaundice.

The Wassermann was positive and the gastric analysis showed 56 degrees of free HCl and a total acid of 75 degrees. The roentgenographic examination showed 60 per cent retention of barium at six hours. The 24-hour plate showed 25 per cent retention. The impression was that the pyloric obstruction was due to ulcer.

On March 5, the patient was placed on a Sippy diet but continued to complain daily of severe abdominal pain. On March 10, early in the morning, the patient suddenly cried out with pain. The intern found marked rigidity of the abdominal wall but did not recognize the condition. Later in the morning the medical resident recorded the following: "The patient is on a very scanty diet, but still has attacks of pain. He is much weaker this A. M. and the pulse is very rapid and weak. There is definite rigidity of the abdomen and complaint of pain on palpation. The leukocytosis is 10,000. Impression: Ruptured ulcer."

Following diagnosis there was further delay in operation as the patient refused permission until his mother arrived. Nine hours following perforation the operative findings were as follows: "A large markedly indurated perforated ulcer is present on the superior surface of the first part of the duodenum. The perforation admits the little finger. There is no local evidence of attempt to wall off. The perforation is closed and a posterior gastro-enterostomy performed. The abdomen is drained suprapubically." Following operation he reacted very feebly and expired on March 11 at 7:15 P. M. No autopsy was obtained.

This patient, then, with an eight year history of chronic dyspepsia and a one year history of typical ulcer syndrome, perforated in the hospital on the fifth day of his Sippy diet. Following perforation, due in part to the delayed recognition of the condition and in part to the patient's own insistence, there was nine hours delay in operation. This delay and the performance of a gastro-enterostomy in the presence of a rapid, weak pulse were factors tending toward an unfavorable outcome. Note the history of daily severe upper abdominal pain.

CASE II.—P.R., a white male, 55 years old, was admitted to the Redford Branch, November 5, 1932, having been struck by an automobile. There was a fracture of the right clavicle and a compound fracture of the right leg. No laboratory examinations were made.

The fractures were treated and the patient was progressing favorably under bed rest and a soft diet. His only complaint up to the morning of November 26 was some pain at the sites of fractures. There was no nausea, vomiting, or gastric distress.

At 5:40 A.M. on November 26 he complained of severe pain in the stomach and side, became nauseated and vomited. He had had codeine Gr. ¼ at 8:00 P.M. and this was repeated at 5:40 A.M.

A progress note on the clinical record describes the findings of ruptured ulcer as being fairly typical. The abdomen showed marked rigidity with great tenderness and the temperature, pulse, and respirations were entirely normal. However, in as much as the patient was not operated upon until 3:35 P.M., it seems reasonable to suppose that the diagnosis in the morning was not quite so obvious.

At operation a large amount of bile stained fluid escaped. A perforation about 1 cm. in diameter was found practically at the pyloric junction on the anterior surface. Simple closure was performed and drainage of the right gutter and pelvis was instituted. The patient left the operating room in poor condition and expired 24 hours later.

While this patient was not on an ulcer regimen, bed rest and soft diet might be expected to treat an existing ulcer kindly. Unfortunately, the record does not show any questioning in regard to previous gastric history. Trauma sustained at the time of injury may be mentioned as a possible causative factor. The nine hour operative delay reduced the chance for recovery.

CASE III.—D.J., a colored male, aged 47 years, was admitted October 18, 1934, complaining of midepigastric pain, persistent hiccough, and generalized weakness. This was his fifth hospitalization in six years, each admission being in the fall, and the complaint being essentially the same each time. Relief was obtained in from 4 to 20 days on these various occasions. A deformed duodenal cap was seen on the third admission, and on the fourth a definite penetrating duodenal ulcer.

On the final admission he said that four days previously he had been awakened by a vague "tight, gnawing" pain to the right of and slightly above the umbilicus. He had been on an ulcer diet since the previous admission but had not had as complete relief as he had formerly enjoyed. For two days previous to admission he had been unable to retain anything. On examination there was a marked icteric tinge to the sclerae, and a moderate degree of muscle splinting in the right upper quadrant of the abdomen, with tenderness. Laboratory findings: Hb., 12.2G. R.B.C., 5,030,000. W.B.C., 9,950. Serology, neg. Icteric index, 16. Urine, neg.

Roentgenography of the G.I. tract October 20 showed a penetrating defect, on the lesser curvature of the pylorus at the duodenal junction, with localized tenderness, and no retention.

Although on a rigid Sippy diet and intravenous injections of synodal from the time of

admission, there was persistence of upper abdominal pain for which a potent sedative or narcotic was administered nearly every day. There was no abdominal rigidity at any time. Operation was advised but refused until October 29, when, following three days of particularly severe pain, he consented and operation was scheduled for the following day.

On the morning of October 30 at 4:30 A.M. the intern was called to see the patient who was in extreme agony. The abdomen was board-like and exquisitely tender. The surgical resident was notified and the patient was taken to the operating room at 5:30 A.M. An ulcer, 2 cm. in diameter, was found situated 5 cm. distal to the pyloric vein. In its central portion there was a perforation .5 cm. in diameter. The abdomen contained a large amount of dark green fluid. Simple closure, jejunostomy and abdominal drainage was done.

Following operation his condition remained quite poor. He continued to have abdominal pain, and some hiccoughing; developed severe infection of the wound and anterior abdominal wall; and, following a gradual decline, expired November 26.

At autopsy there was found severe infection of the abdominal wall, multiple intra-abdominal abscesses between coils of intestine and above the stomach; and two chronic perforating ulcers, one in the duodenum, which had recently been repaired, and one prepyloric.

The long history under treatment indicates a resistant lesion, and the severe and persistent pain suggests an actively penetrating ulcer. A study of the films after autopsy shows that both penetrating ulcers were present at the time of roentgenologic examination. This patient had been on an ulcer regimen for one year. He had been on bed rest and rigid Sippy diet in the hospital for 12 days prior to perforation without much effect upon his severe, persistent, upper abdominal pain. Death occurred in spite of very early and competent operation.

CASE IV.—L.S., a white male, aged 43 years, was admitted November 22, 1934, complaining of constipation for eight days. During an admission in October, 1929, he had complained of epigastric pain before meals relieved by eating and sometimes followed by vomiting. No hematemesis or tarry stools. Roentgenography had shown a penetrating ulcer on the lesser curvature; there was occult and microscopic blood in the stool but no anemia; and, after 16 days on a Sippy diet, he had become symptom free and was discharged.

He was readmitted at this time to the Proctologic service with the diagnosis of carcinoma of the sigmoid. He complained of crampy lower abdominal pain after eating, nausea, and vomiting. He had lost 40 pounds in the last year and 8 pounds in the last ten days.

The positive findings on physical examination were that the patient was emaciated, breath sounds were depressed over the upper third of both lungs with question of fine râles at the right apex on inspiratory cough. Rectal examination showed slight enlargement of the prostate. The abdominal examination was entirely negative. Laboratory findings: Hb., 10.6G. R.B.C., 2,940,000. W.B.C., 11,200. Kline, neg. Urine, neg. Blood in stool.

On November 24, sigmoidoscopy was done, revealing no pathology, although there was old blood in the lower rectum. On November 26, he vomited about 500 cc. of bright red blood, and was accepted on the medical service as a bleeding peptic ulcer. On November 27 the blood picture was Hb., 4.8G., and the R.B.C. 1,800,000. He was treated for this condition by appropriate methods until December 21, a period of three weeks, at which time the Hb. was again 10.4G. with the R.B.C. 3,560,000.

Roentgenography of the G.I. tract on December 19 revealed a large penetrating defect on the lesser curvature midway between the cardia and pylorus and measuring 3 cm. in diameter at the base, and 2 cm. in depth. There was evidence of rigidity of the wall on the lesser curvature above and below the lesion and 90 per cent retention at

the end of six hours. Impression: Large penetrating ulcer of the stomach, probably malignant.

On December 21, it was planned to do a rapid exploration and jejunostomy for feeding while resting the stomach. The operative dictation follows: "There is a large ulcer about the midportion of the lesser curvature, which is about one inch in diameter, including induration. It is quite firm and is sharply limited. There are no regional palpable nodes. It is attached posteriorly. While this could easily be malignancy, on the basis of the gross findings we must call it an ulcer. It is resectable. A jejunostomy was done."

Following operation the patient received nothing by mouth until January 1, 1935, when he was allowed ice chips occasionally. Feedings and medications were given by jejunostomy tube. He continued to complain of some hypogastric pain which was considered due to some fault in jejunal feeding, but was progressing favorably and was up and about the ward. He gained in weight but the amount was not recorded. On January 9, the Hb. was 12.8G. and the R.B.C. 3,800,000.

On January 15, 22 days after operation, the patient appeared rather stuporous when awakened but made no complaint. The previous day he had felt well. At about 10:00 A.M. he seemed so unusually ill that attention was called to his condition. He had complained of no pain and there were no unusual abdominal findings. He was unconscious at noon, and it was thought embolism might have occurred. His temperature rose from normal of the previous day to 104.7°, the pulse to 130 and the respirations to 24. He remained in state of collapse in spite of stimulation and expired at 4:45 P.M.

At autopsy there were about 1,000 cc. of fluid present in the abdominal cavity, and a perforation of the stomach on the lesser curvature midway between the pylorus and cardia was found. The perforation measured about 2 by 4 cm. There was only a slight amount of induration about the perforation. There were a few soft nodes about the greater curvature of the stomach. There was no leakage about the jejunostomy tube.

In this case, one and one-half months following a severe gastric hemorrhage, where an ulcer seems to have existed at least five years, and, in spite of rigid Sippy diet for the first half of this period, plus absolute gastric rest as afforded by jejunostomy during the second half, there was perforation. Another unusual feature is that, in spite of the large size of this ulcer, malignancy could not be demonstrated at autopsy and most of the induration had disappeared. In addition, the shock of perforation was extreme and the ordinary symptoms and signs were absent.

CASE V.—C. H., a white male, aged 53 years, was admitted January 10, 1935, complaining of epigastric pain with vomiting. There had never been any stomach trouble whatsoever until about three weeks prior to admission, when he began to have a dull epigastric ache coming on one half hour following meals. It was relieved somewhat by food, particularly milk, and by soda. He was awakened at about 3:00 A.M. by pain. At times it persisted throughout the day and was occasionally absent for an entire day. During the same period he had vomited six or eight times, always following the evening meal. There was hematemesis one week previous, and the stools had been black. There had been a weight loss of 20 pounds in three weeks.

On examination, the patient was a large framed, heavy set, middle aged man showing slight epigastric tenderness. Laboratory findings: Hb., 9.G. R.B.C., 3,700,000. W.B.C., 9,150. Kline, neg. N.P.N., 37.5. Chlorides, 346. Urine, neg. The gastric analysis showed free HCl in all specimens with a total acidity of 114 degrees, and a trace of occult blood. Stool showed occult and microscopic blood.

On January 8, roentgenologic study of the G.I. tract revealed a large penetrating

defect 3 cm. in diameter at the base, and 1.5 cm. in depth, situated on the lesser curvature 5 cm. above the pylorus. At six hours there was slight retention of barium.

On January 10, the patient was placed on Sippy diet. Three days later, at 10:00 P.M., he experienced a sharp upper abdominal pain which radiated to his left shoulder. He felt faint and dizzy and it was noted that he was in a mild degree of shock. He complained of no extreme pain but rather of a severe dull ache. He was seen by the intern and the medical and surgical residents. The intern noted on the chart that there was no abdominal rigidity but was of the impression that the patient had had a small perforation. At this time the condition suggested a coronary attack. The next morning, leukocyte count was 15,000 with 93 per cent polymorphonuclear cells. There was still no abdominal rigidity, although there was generalized abdominal tenderness with rebound tenderness and questionable obliteration of liver dulness. An electrocardiogram in the morning showed some findings suggestive of coronary thrombosis and therefore helped to delay action. It was repeated in the afternoon and was negative.

Fluoroscopy of the diaphragm was reported as follows: "There is an elevation of the left leaf of the diaphragm which was not present at the time of the G.I. examination. There is marked restriction in the excursion of the left leaf. There is no free air in the peritoneal cavity. In view of the presence of the large penetrating ulcer, we feel that the findings suggest leakage of the ulcer."

The patient had been placed on an Ochsner regimen and did fairly well but that evening when the temperature rose to 100.6°, he was operated upon, about 20 hours after onset of pain. The findings were as follows: "There is no gas or free fluid upon opening the abdomen. There are recent plastic adhesions between the stomach and the liver. On separating these a sloughing area about one and one half inches long and about one inch wide at its widest portion is found on the anterior surface of the stomach next to the lesser curvature. There is no gas or air and careful examination does not disclose any point of actual perforation. It appears that the stomach wall is breaking down over this area, resulting in infection rather than frank perforation. The left side of the greater omentum is drawn up and sutured over the defect in the stomach. There is purulent fluid in the pelvis. A stab wound drain is placed suprapubically."

Following operation, the patient did well for three or four days. His abdomen became distended, however, and he had a partial wound disruption. The T.P.R. remained high. He developed a paralytic ileus and expired January 23, nine days after operation.

At autopsy a purulent peritonitis was found. "On the anterior wall of the stomach 3 cm. proximal to the pylorus, is a large ulcerated area, measuring 3 cm. in length and 5 cm. in width. In the center of this area is a 5 Mm. perforation through the ulcer base. There is a firm ring of induration surrounding this point of perforation on the external surface of the stomach."

Here again we have a quite large gastric ulcer without autopsy evidence of malignancy. The perforation was atypical in that there was no marked abdominal tenderness or rigidity, and this probably because of a dissolution of the ulcer base somewhat gradually, rather than a sudden bursting through. There was also perforation shortly following hemorrhage. The perforation was preceded, however, by almost daily, quite severe, upper abdominal pain of three weeks' duration, and occurred while the patient was on bed rest and Sippy diet.

CASE VI.—H. H., a white male, aged 53 years, was admitted November 16, 1934, complaining of vomiting, abdominal pain, loss of weight and constipation. In March of 1934, the patient began to have attacks of vomiting and severe constipation. He was treated elsewhere for these complaints in April without relief. Treatment consisted of large white pills taken four times a day. Roentgenologic examination at that time showed

what appeared to be a very small lesion on the lesser curvature, which was considered a beginning penetrating ulcer.

In July, 1934, he began having pain about the umbilicus which was of burning type and worse after eating a large meal. At this time he was told he should have an operation on his stomach. From July until admission the vomiting, umbilical pain and loss of weight had become progressively worse. He had lost 70 pounds in one year. There had been no hematemesis but there had been some tarry stools.

Examination showed an elderly, emaciated, white male, with marked sclerosis of the peripheral vessels. The B.P. was 120/80. The abdomen was soft and flat and there was considerable tenderness about the umbilicus. There was a questionable mass in the epigastrium slightly to the left of the midline which was not tender. The liver extended two finger breadths below the costal margin. Laboratory findings: Hb., 10G. R.B.C., 3,540,000. W.B.C., 16,500. Kline, positive. N.P.N., 39. Chlorides, 396. Urine, neg. The gastric analysis showed no free HCl, total acid of 145 degrees, lactic acid, Boas-Oppler bacilli, yeast and occult blood all positive. There was occult and microscopic blood in the stool.

Roentgenography of the G.I. tract on November 21 showed the esophagus slightly dilated. "As the barium enters the cardiac portion, the mucosa appears markedly irregular but there is no abnormality in the contour of the gastric wall. There is an irregular filling defect involving the prepyloric portion and antrum with an irregularity of the wall on the lesser curvature to the midportion of the stomach. There is 95 per cent retention in six hours and 60 per cent at 24 hours. Impression: Carcinoma of the pylorus with questionable involvement of the cardia.

On December 3, he was transferred to the surgical service for operation on the following day. His stomach was not lavaged the evening prior to operation. Although given ten grains of barbital, the patient was unable to sleep, and the intern was called to see him at 4:00 A.M. because of severe pain on the left side of the abdomen. He ordered morphine Gr. ¼, but wrote no note of the patient's condition. In the morning he was seen by the surgical resident, who found some distention of the abdomen but no pain on palpation. The abdomen was doughy and not rigid. He was operated upon at 9:30 A.M., the dictation being as follows: "There is a perforation on the lesser curvature about one and one quarter inches from the pylorus with gastric contents discharging. The perforation was closed satisfactorily and the omentum tacked over it. The extent of the carcinoma and metastasis was so great that a retrocolic gastro-enterostomy could not be done. For that reason, an anterior gastrojejunostomy was necessary. The abdomen was drained through stab wounds. A lymph node was taken from the greater curvature for biopsy."

Following operation his condition remained poor, and he expired on December 7. The pathologic report was medullary adenocarcinoma of a prepyloric lymph node. No autopsy was obtained.

Perforation was again atypical, there being no abdominal tenderness or rigidity. The usual gastric lavage had been omitted the evening prior to operation and so was not a factor in causing perforation. Since admission to the hospital he had been on bed rest and soft or fluid diet.

CASE VII.—J. R., a colored male, aged 43 years, was admitted January 4, 1935, complaining of severe pain in the right upper quadrant, nausea and vomiting. These symptoms had been intermittently present for 12 years. There was relief from soda, vomiting and food. The severity of symptoms had gradually increased during the previous month. No hematemesis, tarry stools, or weight loss prior to admission. Gastric ulcer had been diagnosed by roentgenography at another hospital five years previously.

The patient was a fairly well developed colored male, nauseated and in great pain. The heart was not enlarged, the blood pressure 170/120, and there was an occasional

dropped beat. The abdomen was soft, not distended, no areas of tenderness, no rigidity, and no tumor masses. Laboratory findings: Blood, 1-4-35:Hb., 11G. R.B.C., 3,900,000. W.B.C., 15,200. Kline, negative. N.P.N., 28. Chlorides, 330. CO_2, 55. Calcium, 12.3. Bleeding time, two minutes. Clotting time, four and one half minutes. Icterus index, 4.5. Urine, neg. Gastric analysis showed free HCl, in all specimens, total acid 80 degrees, gross and occult blood positive, W.B.C., five; R.B.C., 40. Stool—occult and microscopic blood. Graham-Cole test showed non-visualization of the G.B.

Roentgenography of the G.I. tract on January 5 showed "a stomach of cascade type with marked hypertrophy of the rugae. Duodenum is visualized with difficulty revealing a persistent deformity of the cap and slight dilatation. There is about 10 per cent retention of barium in the stomach at six hours.

"Impression: Duodenal ulcer with chronic gastritis." (The roentgenologist requested a reexamination.)

He was placed on Sippy diet pending surgical interference. This afforded meager relief from symptoms and on January 16 it was proposed that he be operated on the following day. However, at 8:00 P.M. that evening he had a copious hematemesis of two liters of blood and gastric content. The B.P. dropped to 78/60, and the R.B.C. to 1,410,000 and the Hb. to 3.6G. He showed at this time, no signs of peritoneal irritation. The patient's shock was treated and the scheduled operation cancelled. He was transfused on January 17, and on January 19, each time 500 cc. being transferred by the direct method. Following this his condition became much improved and the Hb. reading was 7.4G. with R.B.C. 2,500,000.

On January 21, he was operated upon with the intention of doing the simplest procedure which would prevent further hemorrhage. There was severe collapse following injection of spinal anesthetic (pantocain). Restorative measures were effective and his condition warranted the rather imperative operation. "The pylorus and duodenum are perfectly normal. There is a very definite new growth involving the lesser curvature for a distance of about two inches, at just about the midportion. There is some adhesion to the pancreas, but it is thought this can be separated. Condition of the patient at present does not warrant a subtotal gastrectomy. Therefore, a regional lymph node along the greater curvature is removed for pathologic examination. The liver is normal."

Following operation the condition of the patient was only fair and on January 22 a small transfusion was given. That night and the next morning the patient complained of severe upper abdominal pain. In the morning there was increase in the degree of distention, tenderness on palpation of the lower abdomen, but no marked rigidity. Perforation as a complication was suggested, but did not seem to be supported by the findings. Check of the blood count showed elevation of W.B.C. to 18,500. He expired at 1:30 P.M. on January 24.

Autopsy report: "There are about 1,000 cc. of dirty fluid in the abdomen. There is a plastic exudate on the small bowel. Upon the anterior surface of the lesser curvature of the stomach, 5 cm. from the pyloric end of the stomach, is a perforation measuring 3 cm. by 1.5 cm. Upon opening the stomach along the greater curvature there is found a large, indurated, punched-out area, measuring 8 cm. by 4 cm. This area is on the posterior surface of the stomach and has the appearance of being malignant. No gross evidence of metastasis." It was only after repeated sections had shown chronic gastric ulcer that an area of definite malignancy was found, thus supporting the diagnosis of a primary chronic gastric ulcer with recent malignant degeneration.

Perforation in this last case followed so soon after operation that it is possible, although we feel not likely, that exploration of the upper abdomen may have been a factor. Perforation took place while the stomach was at rest and following 17 days' treatment on Sippy diet. Again the findings were atypical in that there was no increase in abdominal tenderness or rigidity

and the complication was only suggested because of recent experience. There had also been recent hemorrhage.

In the hope of finding other similar cases reported, the voluminous ulcer literature was gone over by title without success. However, two cases were picked up in going through articles at random. Doubtless others could have been found by a complete search, but the relative scarcity is evident. Singer and Meyer[3] describe a man who entered the hospital because of a known jejunal ulcer causing much pain. After two days of diet and alkali the condition became acute. Perforation here was not recognized at once. By the time the diagnosis was established sufficient time had elapsed to make it seem wise to temporize, especially since the patient's condition was improving. This proved to be good judgment since an abscess formed, and was drained 12 days later with recovery. Dowdle's[4] case is remarkable in that a second ulcer perforated six days following rupture and closure of a first. The catastrophe interrupted a smooth convalescence, and it was thought that leakage at the suture line had occurred. As in our cases the termination was fatal.

A patient in the hospital with a diagnosis established and accepted by nurses as well as doctors, who is more or less regularly having definite pain, may have a perforation which is unrecognized for from a few to several hours, because it is considered to be only a more severe exacerbation of the pain he has been having. Only the typical board-like rigidity of the abdomen may force the diagnosis on the unsuspecting intern. Indeed, one complained of lack of cooperation on the part of the patient because he "would not relax his abdomen" so he could be properly examined. Patients who have recently

RÉSUMÉ OF RELEVANT DATA

Prior to Perforation

Case	Pain	Hem.	In Hospital	Ulcer Regimen
1	71 days	None	7 days	5 days
2	Few hrs.	None	22 days	Soft diet
3	16 days	None	12 days	11 days
4	62 days	50 days	54 days	50 days
5	3½ wks.	10 days	3 days	31 days
6	4½ mos.	Recent	18 days	Fluid diet
7	2 mos.	6 days	18 days	11 days

DIAGNOSIS OF LESION

	Before Perforation	Of Perforation
1.	Yes	4 hrs.—typical
2.	No	8 hrs.—typical
3.	Yes	At once—typical
4.	Yes	Not suspected
5.	Yes	By intern at once—staff 12 hrs.
6.	Yes { Ulcer in March / Ca. in November }	Not suspected
7.	Yes { Ulcer before operation / Ca. after operation }	Considered but rejected

been operated upon are in a more unfortunate position. It is very difficult to separate new symptoms and physical findings with sufficient clarity to warrant immediate reoperation. Fluoroscopic demonstration of air underneath the diaphragm may be the only way in which this can be justified. The condition rapidly progresses to the point where operation is hopeless. It is rather a paradox, but the patient who perforates while in the hospital has less chance of recovery than the one who brings his perforation to the hospital.

COMMENT

(1) The frequently repeated statement that "a bleeding ulcer will not perforate and a perforating ulcer will not bleed" cannot be relied upon. This is particularly true in gastric ulcer.

(2) We cannot feel secure that when we have placed an ulcer case on the most rigid regimen he is immune to perforation.

(3) A grave responsibility must be assumed by the physician or surgeon who does not advise operation for a patient with a known or strongly suspected gastric or duodenal lesion causing persistent pain.

(4) Because the ulcer cases here cited perforated under conditions most favorable for healing, and because in spite of very early operation in one and fairly early in two others, all died with a purulent peritonitis, we may suspect a more virulent organism resident in such ulcers.

(5) Acute perforation may be a complication of carcinoma of the stomach.

(6) Symptoms are apt to be sufficiently atypical or clouded by an associated condition to be inconclusive.

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FACTORS IN THE MORTALITY OF ACUTE APPENDICITIS

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THIS report is based on a study of the cases of acute appendicitis admitted to the University Hospital, Iowa City, Iowa, a State Hospital primarily for indigent patients, which receives its clientele from a radius of over 300 miles. The series includes 1,211 patients admitted during the 15-year period from 1919 to 1934, all of whom were operated upon with the exception of three patients who were moribund on admission.

Because of the voluminous literature on all phases of acute appendicitis, it is not proposed to discuss the factors influencing the mortality but it is desired mainly to give the results of this investigation; for the most part in tabular form.

The reasons for this study were two-fold: first, to determine if our mortality rate has been increasing, as has so frequently been reported elsewhere; and secondly, to determine factors through which we may be able to reduce our present mortality rate.

In a review of this nature the cases necessarily must be subdivided into certain groups. That any such subdivision is not wholly accurate is admitted, as the impression gained by one individual in reviewing a record may vary somewhat from that of another reviewing the same case. Even at the time of operation, there is an individual variation in the interpretation as to the extent of the pathology. This is not so evident in regard to the immediate pathology in the appendix, but is apparent in the diversity of opinion regarding the character of the exudate in the abdominal cavity. What is regarded as an infectious exudate by one surgeon may be called simply a protective exudate by another. In this review the presence of an exudate in the abdominal cavity, even though it be cloudy, is not considered as evidence of a peritonitis. Unless other criteria of peritonitis were present, this exudate has been considered a protective exudate.

The cases have been divided into four groups.

GROUP 1. *Acute Appendicitis, Uncomplicated:* This group includes those simple uncomplicated and nonruptured cases of acute inflammation.

GROUP 2. *Appendiceal Abscess:* If the case presented a definite and well localized abscess beyond the confines of the appendix it is included in this group.

GROUP 3. *Local Peritonitis:* In this group have been placed those cases in which there was obvious gross infection, beyond the confines of the appendix, which was not walled off, but was still quite well localized in the immediate region of the appendix and a spreading or diffuse peritonitis was not present.

GROUP 4. *Spreading Peritonitis:* All the advanced forms of peritonitis are included in this group.

Incidence and Mortality.—In Table I is summarized the incidence of the cases in the four pathologic groups and their mortality rates. The cases are divided into five year periods for comparative analysis.

TABLE I

INCIDENCE AND MORTALITY

In Five Year Periods

	Number Cases		Mortality		Total Mortality
1919–1924					
Acute appendicitis, uncomplicated	143	(68.4%)	0 — 0.0%		
Appendiceal abscess	44		4 — 9.1%		
Local peritonitis	10		0 — 0.0%	22%	
Spreading peritonitis	12		5 — 41.6%		
	209				4.3%
1924–1929					
Acute appendicitis, uncomplicated	172	(65.9%)	0 — 0.0%		
Appendiceal abscess	52		4 — 7.7%		
Local peritonitis	13		0 — 0.0%	29.7%	
Spreading peritonitis	24		11 — 45.8%		
	261				5.7%
1929–1934					
Acute appendicitis, uncomplicated	499	(67.3%)	3 — 0.6%		
Appendiceal abscess	172		15 — 8.7%		
Local peritonitis	25		2 — 8.0%	30%	
Spreading peritonitis	45		19 — 42.2%		
	741				5.3%
Total—15 Years					
Acute appendicitis, uncomplicated	814	(67.2%)	3 — 0.37%		
Appendiceal abscess	268	(22.1%)	23 — 8.6%		
Local peritonitis	48	(4.0%)	2 — 4.2%	28.7%	
Spreading peritonitis	81	(6.7%)	35 — 43.2%		
	1,211				5.2%

Although our total mortality rate is 5.2 per cent, that of the first five year period is 4.3 per cent, an appreciable difference—and yet the incidence of uncomplicated cases does not vary materially in the five year periods. There are two factors which may account for this lower mortality rate in the first five year period. First, in that period, because the surgical staff was small, there were fewer men operating upon these cases. In the past ten years the Junior Staff has been materially increased and the operative responsibility has been spread over a relatively larger group of surgeons. Secondly, there is the factor of the type of incision used. This point will be considered later, so for the present it will be sufficient to say that a McBurney incision was

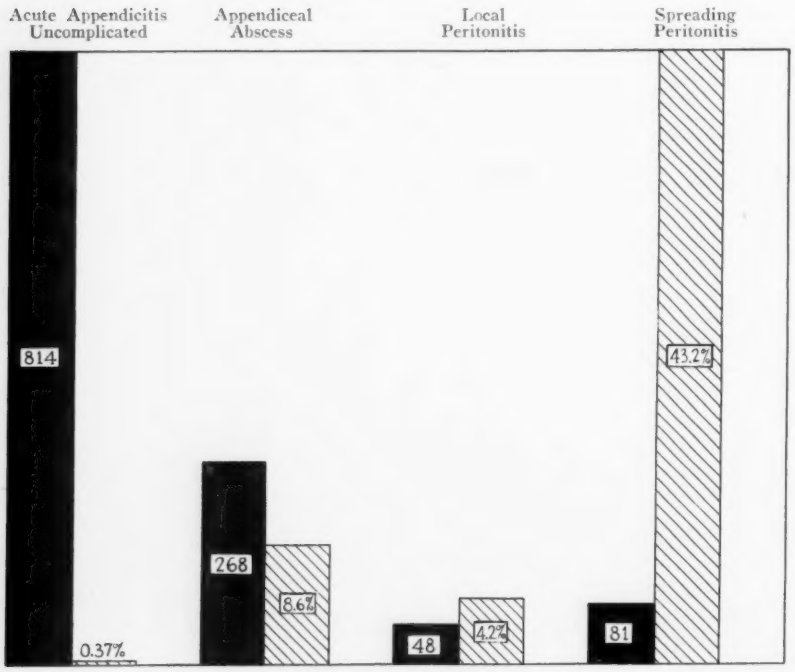
used in a much higher percentage of the cases operated upon between the years 1919 and 1924.

The incidence in the different groups, however, remains fairly constant throughout the 15 years. The proportion of cases that were complicated on admission is extremely high, as approximately one out of every three had progressed beyond the uncomplicated stage.

GRAPH 1

INCIDENCE AND MORTALITY

15 Years—1,211 Cases



Full black column—number of cases.
Black striped column—mortality in per cent.

Age and Mortality.—There is an unusually high mortality in the two extremes of life. In this series there were 200 patients under ten or over 50 years of age. This isolated group accounted for only 16.2 per cent of the total incidence, but 42.9 per cent of all deaths occurred in these patients (Graph 2).

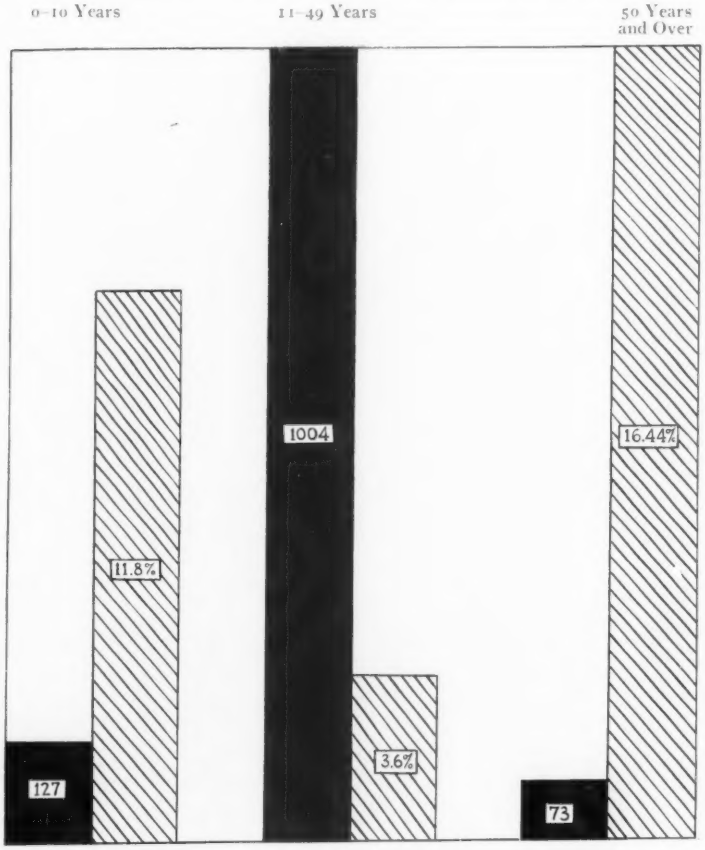
A very striking fact brought out in studying those patients over 50 years of age was the disproportion between the extreme severity of the pathologic change in the appendix and the relatively mild clinical picture presented. Of the 73 cases, only 27 were uncomplicated, while 36 had abscesses and 10 had a spreading peritonitis. Furthermore, of the 27 uncomplicated cases, gangrene or empyema was present in 18, and in several others a markedly inflamed organ was noted. Thus, of the 73 cases, 64 had either perforated or

in all likelihood would have gone on to perforation if operative intervention had not occurred.

That inflammation, once involving the appendix of older people, spreads rapidly, can also be shown. There were 36 patients seen within 50 hours

GRAPH 2

INCIDENCE AND MORTALITY IN THE EXTREMES OF LIFE



Seven cases. No age given.
Full black column—number of cases.
Black striped column—mortality in per cent.

after the onset of their symptoms, and 13 of these were complicated. In the remaining 23 patients, 17 had gangrene or empyema of the appendix. Even in the seven patients whose symptoms had been present under 12 hours, a gangrene or empyema was described in six.

The mortality rate for each group in these elderly patients was:

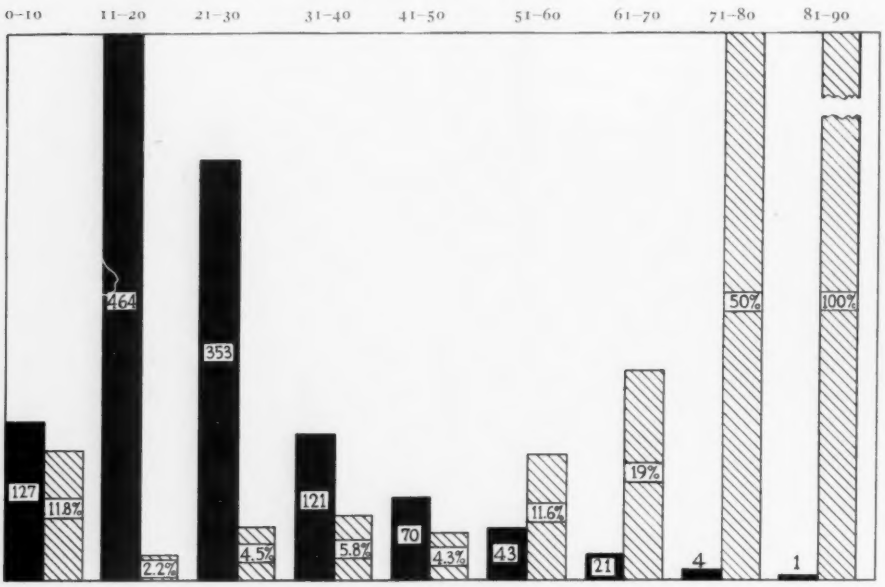
	Cases	Mortality
Acute appendicitis, uncomplicated	27	2 — 7.4%
Appendiceal abscess	36	7 — 19.4%
Spreading peritonitis	10	3 — 30.0%
Totals	73	12 — 16.4%

A critical study was also made in those patients six years of age and under. There were 38 patients in this group with six deaths—a mortality rate of 15.6 per cent. Three of the patients were two years old, six each were three, four and five years of age, and 17 were six years old.

One of the obstacles encountered in these children is the apparent inability or difficulty in making the diagnosis early. As evidence of this is the fact that 19 cases, or just one-half, had had their symptoms for over 72 hours by the time they were admitted to the hospital.

GRAPH 3

AGE INCIDENCE AND MORTALITY



Seven cases. No age given.
Full black column—number of patients in each decade.
Oblique striped column—mortality in each decade (per cent).

In all, pathologically, only 13 were uncomplicated, while 17 had abscesses and eight had a spreading peritonitis. Of the 17 patients with abscesses, all were operated upon (eight drained only) with no deaths. All six fatalities occurred in the eight children with spreading peritonitis. Two of these were moribund on admission and were not operated upon, and one was so ill that simply a drain was placed in the abdominal cavity (Graph 3).

In the 38 cases, a history of previous attacks resembling the present one was obtained in five.

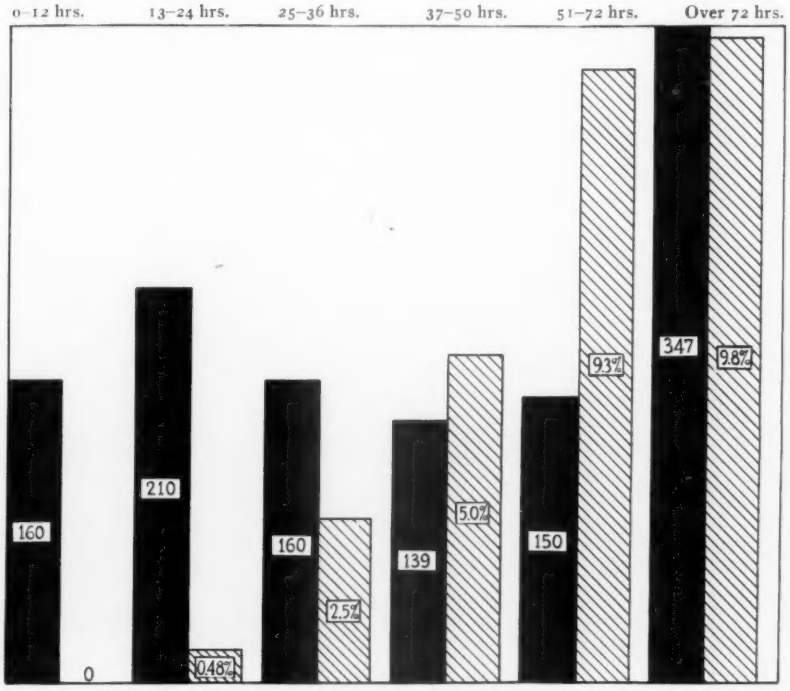
Sex and Mortality.—In this series, males predominated over females, the incidence being 59.7 to 40.3 per cent. The mortality rate for males was 5.4 to 4.9 per cent for females. This difference is explained by the fact that in females 26.8 per cent of the cases were complicated, while in males 36.7 per cent of the cases were complicated.

Duration of Symptoms and Mortality.—The correlation between the dura-

MORTALITY OF ACUTE APPENDICITIS

GRAPH 4

DURATION OF SYMPTOMS AND MORTALITY



Forty-five cases. No time given. Three deaths.
Full black column—number of patients in each period.
Oblique striped column—mortality in each period (per cent).

TABLE II

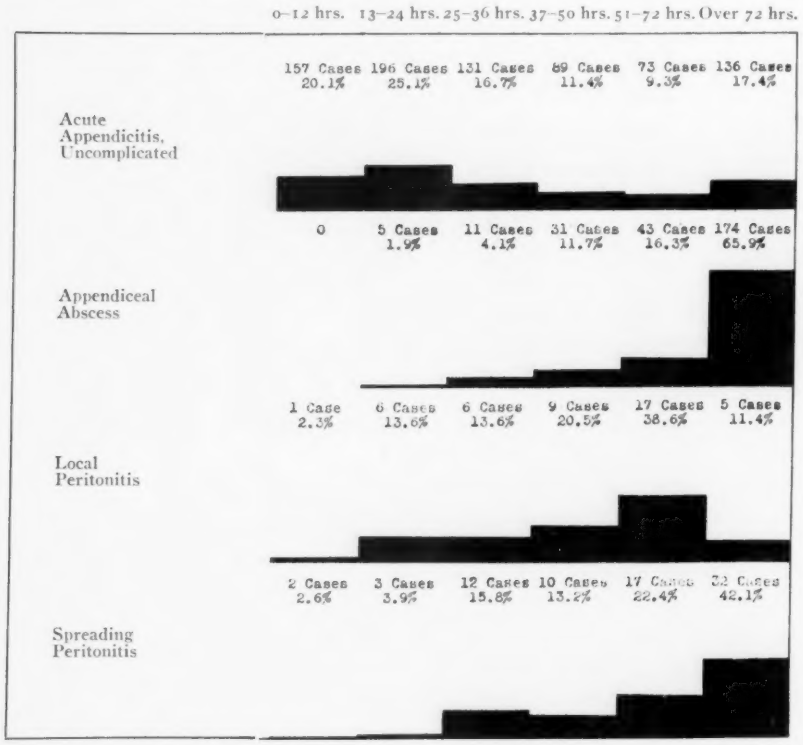
MOST FREQUENT ADDITIONAL DIAGNOSES

	Cases	Mortality
Pulmonary tuberculosis (tuberculous appendix—2)	24	2
Inguinal hernia	10	1
Positive Wassermann reaction (tabes dorsalis—2)	9	1
Pregnancy (one case aborted)	9	
Diabetes mellitus	6	3
Acute upper respiratory infection	4	
Psychosis or mental deterioration	4	
Tuberculosis spondylitis	2	
Fracture	2	
Bronchiectasis	2	
Rheumatic heart disease	2	
Chronic pelvic inflammatory disease	2	
Chronic empyema cavity	2	
Residual paralysis of acute anterior poliomyelitis	2	
Osteo-arthritis	2	
Bronchopneumonia	2	2
Cardiovascular collapse	2	2

tion of symptoms and mortality presents an interesting study (Graph 4). Murphy's dictum, "The earlier the operation the lower the mortality," is at once apparent. Of the 347 cases (28.7 per cent) whose symptoms were present for over 72 hours, 211 were complicated, and the mortality for the whole group was 9.8 per cent. Of the 669 cases (57.4 per cent) in which the disease had been in progress less than 50 hours, there was a mortality rate of only 1.8 per cent.

GRAPH 5

DURATION OF SYMPTOMS AND PATHOLOGY



Forty-five Cases. No time given. Three deaths.

In particular, note: (1) The high incidence of complicated cases before 50 hours. (2) The steady increase in cases developing spreading peritonitis. (3) Sudden drop of cases with local peritonitis after the seventy-second hour. (4) The high incidence of abscesses after the seventy-second hour. (5) The decline in uncomplicated cases after the twenty-fourth hour.

On the other hand, Graph 5 brings out one point which is not sufficiently stressed in the literature: that is, the high incidence of complicated cases developing before the disease is 50 hours old. In this series the number of complicated cases observed in each group before the disease had been in progress for 50 hours is, appendiceal abscess 17.8 per cent; local peritonitis 50.0 per cent, and spreading peritonitis 35.5 per cent.

Additional Diagnoses.—These accompanying conditions are listed in Table

II along with the mortalities that occurred with each disease. A few explanations must necessarily accompany this table.

In the 24 patients with pulmonary tuberculosis, two deaths occurred. At operation one of these patients had an appendiceal abscess and the other had a spreading peritonitis. It is of interest to note that, in the whole group, histologically, only two of the appendices which were removed showed evidence of tuberculosis.

In the one patient with an inguinal hernia who succumbed, the appendix had ruptured in the hernial sac with the formation of an abscess. This patient died of a diffuse peritonitis.

An associated diabetes mellitus exerted a profound influence in the prognosis in this series. There were six cases, and of these four were admitted with a spreading peritonitis. Death occurred in three of the four patients with spreading peritonitis.

Nine patients were admitted with pregnancies varying from four weeks to eight months. There were no maternal deaths and but one case aborted—with a fetal mortality. The pregnancy in this case was of six months' duration and a local peritonitis was found at operation. In one other case drainage of an appendiceal abscess was delayed until the day following delivery.

TABLE III

MOST FREQUENT IMMEDIATE COMPLICATIONS

In 1,148 Patients that Lived

Wound infection and cellulitis	67
Pelvic abscess	41
Acute follicular tonsilitis	17
Abscesses in R.L.Q. and L.L.Q.	11
Abscess site of hyperdermoclysis	10
Fecal fistula (two necessitated operation)	9
Acute intestinal obstruction (one necessitated operation)	9
Atelectasis (right side, 8 cases; left side, 1 case)	9
Thrombophlebitis (left side, 7 cases; right side, 1 case)	8
Pneumonia	7
Pulmonary infarct	6
Subdiaphragmatic abscess	2
Subhepatic abscess	2
Jaundice (probably from septicemia)	2
Psychosis	2

TABLE IV

LATE SEQUELAE

Acute Appendicitis — Uncomplicated	
Acute intestinal obstruction (adhesions)	5
One case had volvulus in addition (resected)	
Incisional hernia	1
	6

TABLE IV—*Continued*

Appendiceal Abscess	
Reformed abscess	12
Acute intestinal obstruction (adhesions)	2
Incisional hernia	2
Acute intestinal obstruction from reformed abscess	1
Incisional hernia and reformed abscess	1
Extraperitoneal abscess with draining sinus	1
Incisional hernia, residual abscess, appendico-sigmoid fistula	1
Acute appendicitis	1
Acute appendicitis with local peritonitis	1
	22
Local Peritonitis	
Acute intestinal obstruction (adhesions)	2
One treated conservatively.	
Spreading Peritonitis	
Acute intestinal obstruction (adhesions)	1
Acute intestinal obstruction from residual abscess	1
Draining sinus—sponge removed twenty-sixth day	1
	3

One case of acute intestinal obstruction (in appendiceal abscess group) died.

TABLE V

CAUSES OF DEATH

63 Cases

Acute Appendicitis — Uncomplicated	
Massive atelectasis	1
Bronchopneumonia	1
Diffuse peritonitis	1
	3
Appendiceal Abscess	
Diffuse peritonitis	8
Bronchopneumonia	3
Bronchopneumonia and diffuse peritonitis	2
Septicemia	1
Septicemia and bronchopneumonia	1
Septicemia and diffuse peritonitis	1
Septicemia and multiple liver abscesses	1
Diffuse peritonitis and acute nephritis	1
Pulmonary embolus	1
Subdiaphragmatic abscess and acute empyema	1
Cardiac failure	1
Acute meningitis (*Staphylococcus aureus*)	1
Acute meningitis, pneumonia and multiple liver abscesses	1
	23
Local Peritonitis	
Diffuse peritonitis	1
Diffuse peritonitis and septicemia	1
	2

TABLE V—*Continued*

Spreading Peritonitis	
Diffuse peritonitis	21
Diffuse peritonitis and septicemia	7
Diffuse peritonitis and bronchopneumonia	2
Diffuse peritonitis and pulmonary edema	2
Diffuse peritonitis, septicemia and bronchopneumonia	1
Spinal anesthesia (peritonitis five days—died on table)	1
	35

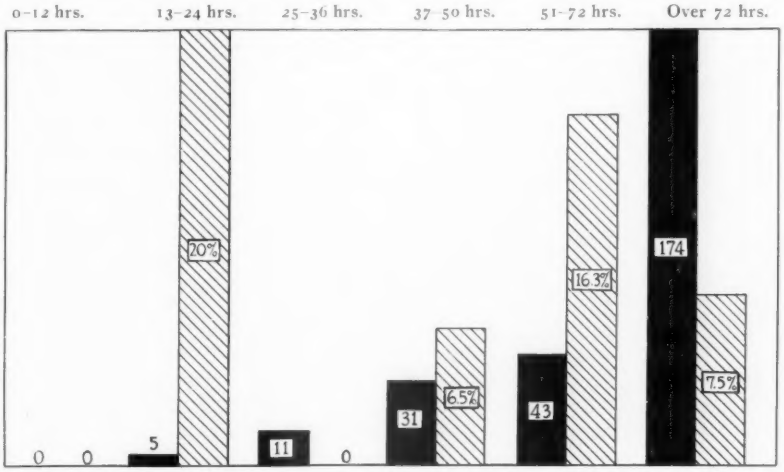
Previous Attacks.—A high incidence of a history of previous attacks was observed in this series. However, the mortality rate in this isolated group was quite low. The incidence and mortality were:

	Incidence	Mortality
Acute appendicitis, uncomplicated	45.7%	0
Appendiceal abscess	33.2%	6.7%
Local peritonitis	33.3%	0
Spreading peritonitis	14.8%	41.7%
Total—1,211 patients		2.2%

Appendiceal Abscess.—A study of the cases with appendiceal abscesses is epitomized in Graph 6. The mortality rate was 8.6 per cent. Of particular significance is the high mortality rate when these patients were operated

GRAPH 6

APPENDICEAL ABSCESS. DURATION OF SYMPTOMS AND MORTALITY IN 268 CASES



Full black column—number of cases. Black striped column—mortality in per cent.
Four cases. No time given. No deaths.

Type of operation.

	Cases	Mortality
Drainage and ectomy	147–54.8%	14–9.5%
Drainage alone	121–45.2%	9–7.4%

upon in that period from 50 to 72 hours after the onset of symptoms. This high rate dropped materially when operation was delayed. The drop in the

mortality rate can be explained rationally on the more adequate walling off that undoubtedly obtains as time progresses. This has led to a change of policy in our treatment of appendiceal abscesses so that now, if an abscess can be diagnosed definitely, operation is delayed until a period when adequate walling off has occurred. Also, we feel that it is advisable to use a McBurney incision for this procedure unless there is some definite contraindication.

Type of Incision.—A study of the type of incision used and the associated mortality reveals some facts worthy of consideration. Either a right rectus or McBurney incision was used in all but 30 cases. Three of these 30 did not undergo operation.

In using a right rectus incision the mortality rate was:

	Cases	Mortality
Acute appendicitis, uncomplicated	514	3 — 0.6%
Appendiceal abscess	207	19 — 9.7%
Local peritonitis	42	2 — 4.8%
Spreading peritonitis	74	30 — 40.5%
Totals	837 — 67.5%	54 — 6.4%

The mortality rate for the McBurney incision was:

	Cases	Mortality
Acute appendicitis, uncomplicated	294	0
Appendiceal abscess	43	1 — 2.3%
Local peritonitis	6	0
Spreading peritonitis	1	1 — 100.0%
Totals	344 — 28.4%	2 — 0.6%

There is a striking difference in these rates which especially is noted in the complicated cases. However, the McBurney incision was not used in a sufficient number of the complicated cases to make the analysis of accurate statistical value, although the marked difference in the two groups cannot be passed over lightly.

In the first five years included in this study the McBurney incision was used in 50.7 per cent of the cases in contradistinction to 20.8 per cent in the last five years. In all probability this is a major factor in the lower mortality rate of the first five year period.

That exploration is limited with the use of a McBurney incision is indicated by the fact that of the appendiceal abscesses operated upon through a McBurney incision the appendix was removed in only 16 cases.

Miscellaneous.—Chills occurred in 3.1 per cent of these cases preoperatively. All of these were single chills. That they are of little value prognostically is indicated by the comparable incidence in each group, *i. e.:*

Acute appendicitis, uncomplicated	3.1%
Appendiceal abscess	3.4%
Local peritonitis	4.2%
Spreading peritonitis	2.5%

All of these patients with single preoperative chills recovered.

Two cases had multiple chills postoperatively. One died and a pylephlebitis with liver abscesses was found at autopsy. The other case clinically had pylephlebitis but recovered.

Intestinal parasites were recovered in 11 of the appendices. For the most part a mild inflammation was present and no deaths occurred. The oxyuris vermicularis was present in nine cases and the ascaris lumbricoides in two.

An adenocarcinoma (carcinoid tumor) was found in addition to the inflammation in two cases. One case was uncomplicated and the other had a local peritonitis. Both recovered.

Discussion.—At the present time the foremost controversial question involving acute appendicitis is whether to treat those cases with a spreading peritonitis conservatively until localization occurs or to operate immediately. After studying this group of patients and those series in which conservative therapy has been used we are unable to conclude that either method is so satisfactory that it can be employed to the total disregard of the other. We feel that the treatment should be individualized to suit the patient rather than to lay down any dogmatic rule.

If the patient is seen relatively early after perforation we know that the result of operative treatment is satisfactory, as evidenced by the low mortality rate in that group with local peritonitis. Also, there are many patients in whom a perforation is suspected clinically but at the time of operation the pathology is uncomplicated. In all probability this is the situation in many of those cases treated conservatively for a peritonitis in whom an abscess never forms.

We feel that even in those cases with an advanced peritonitis immediate operation is indicated as long as the patient is in good condition, thereby eliminating the focus of the infection. The argument is advanced that operation traumatizes the peritoneum before immunity is established, and this is deleterious. Even if this were so the operation can be done with a minimal degree of trauma by using a McBurney incision and spinal anesthesia, and by handling all of the tissues with extreme gentleness.

For the most part, conservative treatment in those cases with a spreading peritonitis, is relegated to patients who are in any degree of shock or who are moribund on admission. The only other group of patients whom we believe should be treated conservatively on admission is that group with appendiceal abscesses whose symptoms have been present for less than 72 hours. In this group the abscesses are not adequately walled off to warrant operative manipulation.

CONCLUSIONS

(1) A cloudy exudate in the peritoneal cavity is oftentimes considered as evidence of peritonitis when in reality it is a protective exudate.

(2) The total mortality rate for acute appendicitis in this hospital is 5.2 per cent.

(3) The mortality rate has increased approximately 1.0 per cent in the past ten years, in all probability as a result of an increase in the number of surgeons operating and the more frequent use of a right rectus incision.

(4) Over two-fifths of all deaths occur in those patients in the two extremes of life.

(5) Murphy's dictum, "The earlier the operation the lower the mortality," is aptly illustrated.

(6) An accompanying diabetes mellitus exerts a profound influence on the prognosis in acute appendicitis.

(7) The mortality rate for appendiceal abscesses is 8.6 per cent. From the results of this study we feel that operation upon these patients should be delayed until adequate walling off has obtained.

(8) Unless there is some definite contraindication, a McBurney incision is recommended for both uncomplicated and complicated cases.

(9) Single preoperative chills are of no prognostic value.

(10) The treatment of cases with a spreading peritonitis should be individualized. For the most part we prefer the operative treatment in these patients unless they show evidence of shock.

THE TREATMENT OF GASEOUS DISTENTION OF THE INTESTINE BY THE INHALATION OF NINETY-FIVE PER CENT OXYGEN*

DESCRIPTION OF AN APPARATUS FOR THE CLINICAL ADMINISTRATION OF HIGH OXYGEN MIXTURES

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THE purpose of this communication is to describe a method for the treatment of intractable distention of the small intestine by the inhalation of 95 per cent oxygen. The principle underlying the use of this agent is based on recent studies in animals[1, 2, 3] which demonstrated, (1) that nitrogen and hydrogen are the major constituents of the gases causing distention, and (2) that the inhalation of pure oxygen effects a substantial reduction in the gas volume of the obstructed small intestine inflated with these gases.

The inhalation of pure oxygen accomplishes this result by preventing the entrance of atmospheric nitrogen into the lungs. The nitrogen in the blood and tissues is consequently rapidly exhaled (60 per cent within one hour).[4] The resulting fall in the partial pressure of this gas in the blood not only prevents its diffusion from the blood into the intestine but at the same time accelerates its diffusion from the intestine into the blood. Our experiments showed that after the inhalation of pure oxygen for 24 hours the volume of nitrogen originally injected into the small intestine obstructed at the pylorus and ileocecal valve was reduced approximately 62 per cent in comparison with an absorption of some 10 per cent when air is breathed.

When the small intestine was inflated with hydrogen, the residual gas volume was likewise found to be considerably smaller in an animal breathing oxygen than in one breathing air, although the difference between the two was not nearly so great as in the case of nitrogen. The beneficial effects of oxygen on the intestine distended with hydrogen were found to be due not to any increase in the rate of diffusion of hydrogen from the intestine into the blood, but to the fact that the nitrogen which ordinarily diffuses into the intestine in considerable quantity from the blood has been eliminated and is not available during the period of oxygen inhalation.

The decompressing action of oxygen was however shown to be ineffective for the distended stomach, apparently because the surface area available for absorption is very much less than that of the small intestine.[3] Since the

* The expenses of this investigation were provided by the DeLamar Mobile Research Fund, Harvard Medical School.

stomach, like the unobstructed colon, can be readily deflated by a rubber tube, oxygen should find its usefulness restricted primarily to distention of the small intestine.

It is clear from the foregoing that the closer to 100 per cent the concentration of inspired oxygen the more effective it should be in decreasing the volume of distending gases. In animals 70 per cent oxygen does not reduce the gas volume sufficiently to be of practical value. The oxygen tents in current use do not provide a concentration higher than 70 per cent even with an extravagant supply of oxygen. We have therefore constructed a modified Barach tent (Figs. 1 and 2) which will yield an oxygen concentration of at least 95 per cent for the treatment of obstinate distention.*

The essential change consists in replacing the ordinary tent by a small rubberized canvas helmet, the bottom of which is closed by a very soft pliable rubber diaphragm with a circular defect (diameter three inches) to accommodate the patient's neck snugly and without discomfort. A closed circuit is thereby created which is sufficiently tight and small in volume so that a concentration of oxygen of about 95 per cent or over is rapidly attained.

To reach this percentage it is, of course, necessary to eliminate the room air present in the system as well as the nitrogen exhaled by the patient. All of the lung nitrogen is removed within the first three to five minutes of pure oxygen administration.[4] At the outset, oxygen is delivered at the rate of 12 liters per minute. By means of a two-way valve on the delivery side of the circuit the system is washed out for about 20 seconds several times during the first 15 minutes. During this washing-out period the hood, except for its rigid roof, collapses gently about the patient's head; but upon closure of the valve the hood reinflates rapidly. Following these preliminary maneuvers the rate of flow of oxygen is reduced to from four to five liters per minute. Gas samples obtained from a sampling jet on the return side of the circuit show oxygen percentages varying from 92 to 98 per cent within 15 to 20 minutes after starting. Now and then during the course of the administration of oxygen the apparatus is washed out and replaced by pure oxygen in order to get rid of the small amounts of nitrogen which continue to be eliminated from the blood, tissues and intestine.

The hood is suspended over the patient's head and can be accommodated to any position, although the sitting position is most convenient. Such a hood provides ample elasticity and volume to permit full respiratory excursions without the restricted feeling one experiences in the rigid helmet devised by Benedict[5] for short periods of administration of pure oxygen. In contrast to the ordinary tent, the subject's body is accessible for nursing purposes and for physical examination of the chest, without losing the benefits of oxygen therapy during these manipulations. Subjectively there is an absence of the imprisoned feeling of the ordinary tent which is objectionable to many

* The details of the apparatus were worked out in collaboration with the Warren E. Collins Company of Boston, who constructed the machine as illustrated.

patients. Three celluloid windows provide for ample vision and patients can hear and be heard readily without having to open the tent. A sleeve is provided below the front window for feeding purposes, but it is much simpler

FIG. 1.—Photograph of apparatus for the clinical administration of 95 per cent oxygen.

to deliver food and drink through the opening in the diaphragm around the neck. The resulting inflow of room air is not a serious disadvantage because the entire system can be washed out and pure oxygen replaced within a few minutes.

The rest of the unit is mounted on the same stand as the hood and consists of a motor blower which sucks the air out of the hood and then drives it through two insulated cans filled with ice. One of the cans also contains a tray of soda lime for absorption of CO_2. The air together with additional oxygen from the tank is then delivered through a flattened slit-mouthed nozzle into the hood and is directed downward in front of the subject's face. It is then sucked back through a perforated tube in the top of

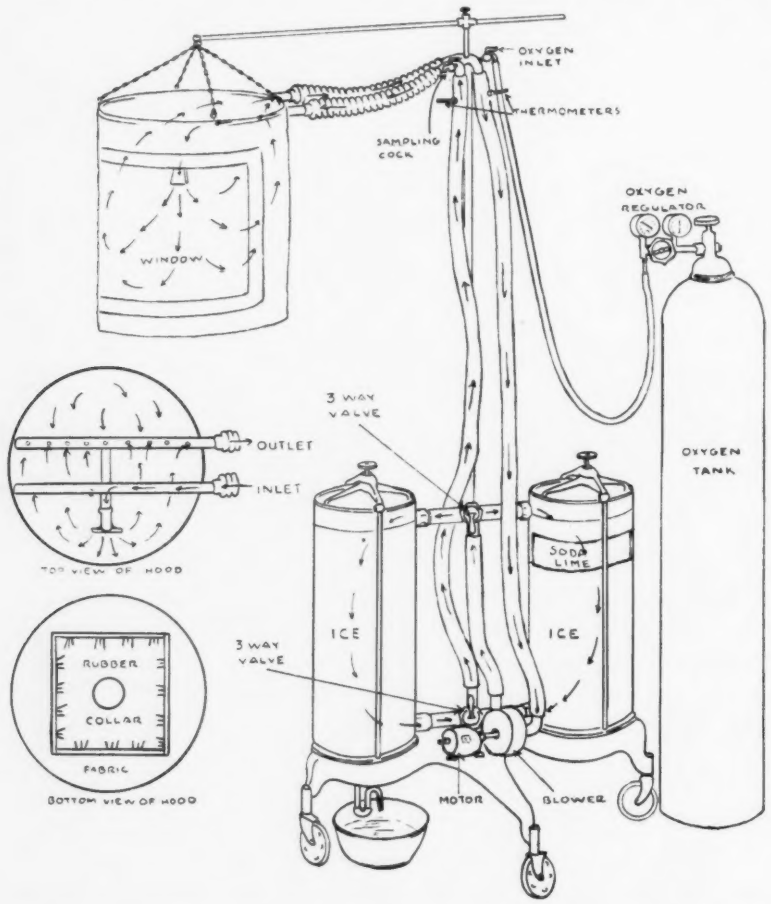

FIG. 2.—Diagrammatic sketch of apparatus for the clinical administration of 95 per cent oxygen.

the hood. Thermometers on the intake and return tubes afford a measure of the cooling efficiency of the system. With a normal human subject in the helmet and a room temperature of 24°C the oxygen enters the hood at a temperature of 15°C to 16°C. When the cooling is inadequate the temperature difference lessens, thus indicating the need for ice replacement. Either ice-can may be refilled, without interrupting the oxygen flow, by shunting the can to be filled out of the circuit by means of valves which

direct the gas flow through both cans or either one, as desired. Water drains out of the bottom of either can through a siphon outlet common to both. A resistance box on the stand controls the intensity of the blast from the blower to meet the subject's preference as to ventilating speed. If for any reason the helmet becomes uncomfortable it can be readily removed, even by the patient. The operating cost of this apparatus is less than that of the ordinary tent because of the smaller volume of oxygen necessary to maintain the desired concentration.

The reduction of the gas volume in the small intestine of animals obtained by this apparatus* compares favorably with the results in previous experiments[3] in which commercially pure oxygen was supplied by a tracheal cannula. Thus the decrease in gas volume in the small intestine by the tracheal cannula method averaged 40 per cent after 12 hours and 57.5 per cent after 24 hours. With this apparatus the results in two 12-hour experiments were 60 and 66 per cent and in two 24-hour experiments 63 and 70 per cent.

Due cognizance must be taken of the toxic properties of high concentrations of oxygen before submitting patients to this agent. We shall deal with this aspect of the subject more fully in a subsequent clinical report. In passing it may be worth summarizing briefly what data is at hand in this connection. Animals can breathe 70 per cent oxygen indefinitely, but higher concentrations eventually produce pulmonary edema. No apparent harm however results from pure oxygen if given to animals for 16 out of every 24 hours for many days.[6] Healthy men can breathe pure oxygen for at least four hours without toxic symptoms.[4] Sayres[7] has exposed men to 100 per cent oxygen for eight hours continuously with impunity. Evans and Dushordwe[8] state that they have given pure oxygen to patients with cardiac or pulmonary disease for 24 hours or longer with nothing but benefit. Since they employ a mask and allow feeding at the same time it may be doubted that the patients receive pure oxygen continuously.

Our clinical experience to date with the use of the apparatus described above is limited to five cases.

CASE REPORTS

CASE I.—A male, aged 65, entered the hospital complaining of generalized abdominal pain of three days' duration. The history was altogether unreliable owing to the confused mental status of the patient. For two years he had been troubled with nocturia and straining during micturition. In spite of repeated enemata he had passed no flatus during the last 24 hours. For the past 12 hours he had vomited a dark brown fluid repeatedly.

Physical examination revealed a dehydrated male with peripheral vascular collapse. The abdomen was severely distended, tense, generally tender, tympanitic everywhere except for dulness to percussion in the flanks and lower abdomen. A fluid wave was present. Red blood cell count 3,600,000, hemoglobin, 70 per cent (T), white blood cell count 20,300, blood nonprotein nitrogen 109, blood chlorides 446. A roentgenologic interpretation of a flat film of the abdomen was as follows: "The small bowel, especially in the left upper quadrant appears distended and shows numerous fluid levels. There is also some disten-

* In the animal experiments we used instead of the helmet a water sealed box supplied with a water valve to allow the escape of excess gas.

tion of the large bowel." Immediate laparotomy through a lower right rectus incision revealed a generalized peritonitis with large quantities of free fluid. In the field of operation the intestine was not distended. Immediately after operation the patient was placed in the helmet and he breathed 95 per cent oxygen until his death 14 hours after operation.

Autopsy revealed an obstructing median lobe of the prostate with a ruptured diverticulum in the dome of the bladder, and generalized peritonitis with over a liter of purulent fluid. Fibrous adhesions were found everywhere among the organs in the peritoneal cavity but there was no evidence of mechanical obstruction. *The entire small intestine was collapsed and contained no gas.* There was no edema, atelectasis or consolidation of the lungs, but there was slight congestion of both bases. The bronchial tree contained a small amount of secretion but no blood. The pulmonary vessels were normal. Microscopic examination of the lungs showed no hemorrhagic extravasation. The alveolar walls were not thickened. A few arterioles contained red cells in clumps resembling small thrombi. The rest of the autopsy was irrelevant.

COMMENT.—These findings indicate that there was no damage to the lungs as a result of 14 hours of continous oxygen inhalation. The evidence in this instance as to the influence of pure oxygen inhalations on the degree of distention is somewhat equivocal, but it is certainly unusual to find the entire small intestine collapsed in cases of generalized peritonitis.

CASE II.—A male, aged 72, was admitted to the hospital with a diagnosis of coronary thrombosis, three weeks after the onset of severe chest pain associated with paroxysmal nocturnal dyspnea, weakness and fever. For four days preceding entry there had been no bowel evacuation. On admission the patient was slightly cyanotic, had a blood pressure of 110/90 and was moderately distended. There were moist râles at the bases of both lungs and he was thought to be developing a bronchopneumonia. The distention increased in spite of enemata, dry heat and turpentine stupes to the abdomen. There was occasional peristalsis but without appreciable expulsion of flatus. Two days after admission the patient was placed in the helmet and 95 per cent oxygen administered for a period of nine and one-half hours with a one-half hour interval outside the helmet.

During the period of oxygen administration the cyanosis disappeared, the pulse was slowed from 92 to 82, the blood pressure remained essentially the same, and the white blood cell count fluctuated from 11,000 to 13,000. *The abdominal girth measurements decreased some six cm.* Flatus was expelled in small amounts from time to time. Three hours after the oxygen was stopped the patient deflated spontaneously and the girth of the abdomen decreased to nearly normal. The patient remained comfortable and the lungs cleared up entirely in the next few days. Five days later the distention recurred but was relieved by ordinary procedures. The patient left the hospital much improved 17 days after entry.

COMMENT.—In brief, this case is one in which a patient with acute coronary disease breathed 95 per cent oxygen for nine hours with no demonstrable deleterious effect. It is by no means conclusive of the value of 95 per cent oxygen for intestinal distention inasmuch as the greater degree of deflation occurred several hours after stopping the inhalation of oxygen. But it is not ureasonable to assume that a severely distended intestine, incapable of peristaltic activity by virtue of the overstretching of its muscle, may be restored to effective peristaltic activity by decreasing its gas volume sufficiently to enable the muscle to recover its tone. Thus it is possible that the oxygen inhalation, having facilitated a significant absorption of nitrogen

via the blood (as evidenced by the six cm. decrease in abdominal girth), made it possible for the intestine to recover its tone and so to complete its deflation. Additional evidence in favor of this assumption is given in the following case history.

CASE III.—A girl, aged six, 72 hours before admission had a severe chill followed by intermittent fever, anorexia, constipation, and oliguria. She had recently recovered from a cold. Shortly before entry she vomited and began to have diarrhea with rectal tenesmus. On admission the child was extremely toxic and pale and was complaining of generalized abdominal pain.

Physical examination was not remarkable except for the abdomen which showed a board-like rigidity and diffuse tenderness, especially in both lower quadrants. The temperature fluctuated between 101°F and 105°F, the pulse from 120 to 140, and the respirations 20. The urine was negative, the red blood cell count was 3,250,000, hemoglobin 65 per cent (T) and the white blood cell count was 22,500 with a differential of 88 per cent polymorphonuclear leukocytes, of which 16 per cent were band forms. Blood and stool cultures were negative. For six days the patient continued to have diarrhea. The abdomen would alternately soften and become rigid. In spite of the fact that she passed flatus freely the abdomen gradually became more distended. On the sixth day after entry the child was slightly cyanotic, respirations became labored and the abdominal distention and rigidity increased. She was put into the helmet and 95 per cent oxygen administered. The following table gives the measurements of the abdominal girth at three levels before, during and after the period of oxygen therapy (Table I).

TABLE I

Abdominal Girth in Centimeters

Date	Hour	At costal margin	At umbilicus	Halfway between umbilicus and symphysis pubis	Remarks	
March 22, '35	5:30 P.M.	66	63.5	64.5		
	5:30 P.M.	Oxygen helmet applied *			No flatus passed during this period.	
	8:30 P.M.	64	64	62		
	11:30 P.M.	63	61.5	61		
March 23, '35	2:30 A.M.	63	61	59.5	2:25	Expelled large amount of flatus.
					3:15	Expelled large amount of flatus and had large stool.
					3:25	Expelled large amount of flatus.
					4:30	Expelling flatus freely.
	5:30 A.M.	61	59	58		
	8:30 A.M.	61	59	58		
	11:30 A.M.	61	60	59	11:30	Abdomen much less distended though not soft.
	2:30 P.M.	60	59	58		
	6:00 P.M.	Oxygen discontinued				
March 24, '35	7:30 A.M.	61	59.5	58		
	5:30 P.M.	63	61	59		
March 25, '35	7:30 A.M.	60.5	59	57		

* From 5:30 P.M. March 22 to 6 P.M. March 23 the helmet was removed every four to six hours for about one-half hour. Total time of exposure to 95 per cent oxygen was 20 hours.

During the period in the helmet the pulse dropped from 130 to about 110, an effect commonly observed when high oxygen mixtures are breathed. The cyanosis disappeared. Shaw and Behnke[4] have noticed an occasional instance of an increase in the white blood cell count, but none occurred in this instance.

The child proceeded to improve rapidly within the next 24 hours and distention did not recur. The diarrhea stopped. Three days later she was well on the road to recovery. At present she shows some induration in the pouch of Douglas and seems to be localizing a small pelvic abscess. The abdomen is flat, and not tender; the appetite and bowel function are practically normal.

COMMENT.—In this patient moderate distention had been present from the time of entry until 36 hours preceding oxygen administration when it became severe. Throughout this period the child had been expelling flatus now and then in small amounts but not sufficient to prevent increasing distention. It will be noted in Table I that for the first eight hours of oxygen inhalation no flatus was expelled per rectum or regurgitated by mouth, but the abdominal girth was substantially reduced. After this, there were frequent discharges of large amounts of flatus so that an effective decompression of the intestine occurred within 15 hours after beginning the oxygen inhalation. Presumably the oxygen had accomplished sufficient shrinking of the bowel by the absorption of nitrogen via the blood so that the intestine (which was undoubtedly in an irritable state as evidenced by the severe diarrhea) was able to complete the deflation by peristalsis.

The prompt and complete recovery which followed almost immediately after 20 hours of breathing 95 per cent oxygen without subjective or objective signs of damage to the brain or lungs constitutes evidence that either the toxic properties of high concentrations of oxygen do not assert themselves until after such an interval or that they can be minimized or avoided by intermittent administration.

In the animal experiments already referred to it was noted that a substantial decrease amounting to over 40 per cent of the initial distending volume was observed after 12 hours of intermittent oxygen administration. In the preceding case a significant decrease was noted within eight hours. In the following case an appreciable decrease was observed within six hours. During this time there was no belching or discharge of flatus by rectum so that the shrinkage in distending volume can be attributed to increased absorption via the blood as a result of the use of oxygen.

CASE IV.—A male, aged 46, house painter by occupation, entered the hospital complaining of generalized abdominal pain and constipation of three days' duration. The pain was characteristic of intestinal colic and had been associated with much retching and occasional vomiting. For three days, in spite of repeated enemata and magnesium sulphate by mouth, he had passed nothing by rectum and gradually became markedly distended. Examination on admission revealed nothing remarkable except pulmonary emphysema and a distended abdomen which was tympanitic throughout to percussion. There was no audible peristalsis. He was suspected of having lead colic. The urine was found to contain lead. Pending a roentgenologic study of the colon the following morning to rule out a large bowel obstruction, he was given 95 per cent oxygen by the helmet (Table II).

TABLE II

Time	Conc. of O_2 in the helmet	Pulse rate	Blood pressure	White blood cell count	Circumference of abdomen in cm. at: Costal margin	Umbilicus	Midway between umb. and symph. pubis
10:40 P.M.	Oxygen started						
10:40 P.M.	90%	76	170/120	10,000	95.5	97.25	95.5
11:30 P.M.	94	76	140/86	..	93.0	98.0	96.5
1:30 A.M.	92	88	160/90	..	95.0	96.0	94.5
2:30 A.M.	99	88	140/90	18,950	93.0	96.0	95.0
4:30 A.M.	98	76	130/86	..	92.0	95.5	92.
7:30 A.M.	94	80	140/90	15,800	92.0	95.	91.0
8:30 A.M.	Oxygen discontinued						
9:30 A.M.	Barium enema; roentgenologic findings negative.						

The patient was by no means completely relieved of distention after ten hours of oxygen therapy, but there was no doubt that the abdomen had been considerably reduced in size within six hours and that distention had been considerably relieved in spite of the fact that no gas was passed by rectum during this period. Oxygen was discontinued during the following day until evening when after another six hours in the helmet an enema resulted in practically complete deflation.

CASE V.—A girl of seven was admitted with bilateral bronchopneumonia and empyema due to a streptococcus infection. She was extremely dyspneic and cyanotic on admission and seemed almost moribund. The abdomen was markedly distended. She was placed in a Barach-Collins oxygen tent for 24 hours but the cyanosis did not appear. Because of this and because there was a well marked abdominal distention which did not yield to hot stupes and enemata she was given 95 per cent oxygen by the helmet. The cyanosis was replaced by a pink color in the lips and fingers. Without removing the helmet she was placed on her side and a left trocar thoracotomy was done. During the following six hours the pulse dropped from 170 to 156, but the respiratory rate remained unchanged at 68. Oxygen was continued for 15 hours, with three interruptions outside of the helmet for periods of 30, ten and five minutes. At the end of this time she expelled a large amount of flatus and the abdomen became soft.

Oxygen was continued, with occasional interruptions totalling two hours, for the next eight hours because intense cyanosis returned as soon as she was removed from the helmet. The following day mild distention recurred, but for the preceding 12 hours she had refused to stay in the helmet except for periods too short to be effective for deflation purposes. The pulse had meanwhile dropped to an average of 140, and the white blood cell count fell from 38,000 to 18,400. These changes were in all probability due to surgical drainage of the left pleural cavity. A right trocar thoracotomy was then done. The child in the course of the subsequent six weeks improved very considerably and is now in the convalescent stage.

COMMENT.—In this patient the dangerously low vital capacity due to the pulmonary infection was threatened still more by abdominal distention. The ordinary oxygen tent had failed to relieve the cyanosis and the usual measures for the relief of the distention had likewise proved ineffective. By the use

of 95 per cent oxygen the cyanosis was dispelled and, as in Cases II and III, an adequate deflation of the intestine was accomplished.

It is impossible to affirm or deny the occurrence of toxic effects of 95 per cent oxygen in this child. However, the pulmonary edema or hemorrhage and the cerebral effects attributable to oxygen poisoning were not observed.

Discussion.—There are a few recorded observations on the effect of 95 per cent oxygen on abdominal distention. Thus Evans and Dushordwe[8] incidentally observed that their pneumonia patients who were given pure oxygen by nasal catheter were relieved of their distention within 36 to 48 hours. They assume that anoxemia causes distention and that the relief of the anoxemia restores intestinal muscle tonus with resulting deflation. By this hypothesis oxygen must be considered only indirectly responsible for the relief of the distention and of value only in virtue of the existence of anoxemia.

W. T. McConnell and R. L. McCormack[9] report 16 cases of distention following cesarean section which were relieved by 95 per cent oxygen and five per cent carbon dioxide. They state that "the distention is due to shock, paralysis of the splanchnic nervous system, loss of smooth muscle tone and decrease in local circulation. An increase in carbon dioxide tension has long been known to stimulate smooth muscle activity. Therefore, one would expect the administration of carbon dioxide and oxygen to stimulate peristalsis and restore normal cellular function." Apparently the authors considered carbon dioxide the chief agent responsible for the relief of the distention, but it is not clear what rôle 95 per cent oxygen played. When one notes further that they used this gas mixture three or four times for five-minute periods every three to four hours, it is difficult to see how the use of 95 per cent oxygen for a total of some 20 minutes in all could have had any important bearing on the results observed.

We have already referred to the experimental evidence showing that the inhalation of 95 per cent oxygen causes a much more rapid deflation of distended intestine than occurs when air is breathed. Since this observation was made on the mechanically obstructed intestine the favorable effect of oxygen in these experiments cannot be attributed to the possible effect of oxygen on muscle tonus except insofar as increased muscle tonus results in increased intra-intestinal pressure with resulting increase in the speed of absorption of intestinal gases. That this can be of nothing more than minor significance however is clear from the evidence we have presented in a previous report.[3] In the unobstructed intestine it is not safe to assume that oxygen aids in the deflation by an effect on muscle tonus until well controlled observations showing such an effect are available.

The oxygen saturation of the blood has little or nothing to do with the effect of breathing pure oxygen on gaseous distention of the intestine. Our experimental observations demonstrate that the effect of oxygen depends primarily on the laws governing gaseous exchange across a semipermeable

membrane and lies in the fact that *by excluding nitrogen from the inspired air*, the nitrogen in the blood and tissues is exhaled and in consequence the nitrogen in the intestine diffuses out of the intestine into the blood much more rapidly than occurs when air is breathed. At the same time nitrogen which ordinarily diffuses from the blood into a distended intestine in large quantities is no longer available for this purpose.

That this mechanism operates in man is apparent from Cases III and IV in which a sizable decrease in abdominal girth was observed after a number of hours of inhalation of 95 per cent oxygen and *before any gas was passed by rectum*. None of the patients included in this report were victims of mechanical obstruction and all of them eventually completed the deflation spontaneously. Their failure to do so for several days preceding the administration of oxygen with all the usual measures for the treatment of distention suggests that the oxygen permitted the unobstructed but overstretched intestine to recover effective peristaltic activity simply by accomplishing a partial shrinking of the bowel lumen.

One may infer that the deflation attributed to oxygen in the five patients reported might well have occurred without its use. This cannot be denied. On the other hand the fact that the distention had persisted in each case for at least several days in spite of intensive treatment and that it was effectively disposed of within a reasonable period after the initiation of oxygen therapy makes the correlation seem more than coincidental. The significance of the oxygen is not minimized by the observation that a large fraction of the distending gases was evacuated by peristalsis. That eventuality should indeed be anticipated in any case of distention of functional origin once the intestine has been relieved of the mechanical disadvantage of overdistention. It is precisely this function which we attribute to oxygen, for in each of the five cases reported the distention remained intractable to the ordinary therapeutic measures and yielded only after oxygen had been observed to decrease the degree of abdominal distention without a concomitant discharge of flatus (Cases I, III, and IV) or with only a negligible discharge (Cases II and V).

There should therefore be no need in the average case of ileus to rely on the administration of oxygen to complete the deflation except perhaps in instances of mechanical obstruction in which operation may be contraindicated, although this will admittedly be a rare circumstance.

Oxygen may prove quite useful in distended patients during a period when the surgeon is not certain whether he is dealing with a mechanical or functional ileus. In a recent experience of this type oxygen was given with the usual result of a decrease in abdominal girth, following which enterostomy was done for mechanical obstruction. At operation it was noted that most of the patient's residual distention was due to large amounts of intra-intestinal and intraperitoneal transudate.

In resorting to the use of 95 per cent oxygen for the treatment of distention we have not ignored the possible dangers involved. The available

evidence indicates that when it is given intermittently its toxic properties can be avoided. Our experimental data has demonstrated that intervals of respite in room air for one-half to one hour following three or four-hour periods in the helmet can be allowed without vitiating the effect of the oxygen already inhaled. Since the needs of the average patient will in any case require occasional interruptions and periods of rest outside the helmet the intermittent administration of oxygen proves to be more practical and at the same time safer than continuous administration. We have observed no untoward respiratory or cerebral effects in either man or animals from oxygen employed in this way for periods lasting as long as 24 hours, which is well within the limits necessary to accomplish the desired result.

The length of time necessary to effect a substantial improvement by oxygen cannot be predicted in any given instance. In the five cases cited the shortest period required was six hours. Since oxygen does not absorb gases from the stomach in any appreciable volume the use of the stomach tube to obviate the entrance of these gases into the intestine is a valuable procedure preliminary to the administration of oxygen and should shorten the time of sojourn in the helmet. Once in the helmet the patient can swallow only oxygen, a gas of no consequence in intestinal distention.[2, 3] The selection of foods which are minimal gas producers[2] will, by preventing the accumulation of hydrogen in the gut, also decrease the period of time necessary for a successful issue.

Although our objective in devising this method is the treatment of obstinate distention such as may be encountered postoperatively, or in peritonitis, or perhaps in cases of suspected mechanical obstruction in which surgical relief is being withheld for one reason or another, its usefulness is by no means restricted to this purpose. Other possible indications are the following: (1) after operations on the gastro-intestinal tract when tension on suture lines must be avoided 95 per cent oxygen may serve as a prophylactic against the occurrence of distention because nitrogen, which is the chief constituent of the distending gases, is thereby excluded from the patient's environment. (2) The apparatus is conveniently applicable for the relief of anoxemia in cardiac and respiratory disorders. Indeed in instances in which anoxemia and distention coexist, a double purpose would be served. (3) Ninety-five per cent oxygen may find a possible usefulness in mediastinal emphysema. We have not as yet encountered a case for trial of the method but on theoretical grounds it may be worth a trial because the absorption of nitrogen from the soft tissues is accelerated by breathing pure oxygen. A recent investigation has established this observation beyond peradventure.[10] (4) Finally, the method may be of value in patients suffering with severe or persistent headache following encephalography. This will be the subject of a forthcoming report.

CONCLUSIONS

(1) Experimental evidence has been obtained which indicates that the

inhalation of 95 per cent oxygen may be effective for the relief of gaseous distention of the intestine.

(2) The inhalation of 95 per cent oxygen is therefore suggested as an agent for the treatment of intestinal distention when the usual methods available for this purpose are either contra-indicated or have proved unavailing.

(3) A safe, practical and economical method for the clinical administration of 95 per cent oxygen for this purpose is described. When properly used the dangers of oxygen poisoning can be avoided.

(4) Five patients with varying disorders, all victims of severe distention, were exposed to 95 per cent oxygen by means of this apparatus for periods varying from eight to 24 hours. The results do not conclusively demonstrate the clinical value of the method, but they are sufficiently gratifying to justify this preliminary report and to encourage further trial.

(5) Indications, aside from intestinal distention, for the use of the method are suggested.

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CYST OF WOLFFIAN ORIGIN WITH APLASIA OF KIDNEY AND URETER, HYDRONEPHROSIS AND UNDESCENDED TESTICLE

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CYSTS originating in the remains of the wolffian body are distinctly rare or relatively common depending upon the number of types one is willing to admit to this classification. Most authors, Ewing[4] for example, apparently accord the wolffian apparatus only a small rôle in the genesis of tumors, while others believe that it is the source of all retroperitoneal cystic tumors which do not demonstratively arise from one or other of the retroperitoneal organs. Some authors go even farther and include pseudomucinous cysts of the ovary (Monnier,[13] Portes[19]), certain tumors of the testis (Ployé,[18] Monserrat[14]), and cysts of the vagina (Lordy). Because of this confusion and because of the scarcity of references in the American literature, it is believed worthwhile to report all observed cases.

The two most recent comprehensive reviews in English are those of Maury[12] and of Hinman.[7] These authors together collected 44 cases, including one each of their own, and the facts which they assembled relative to wolffian cysts are briefly as follows:

Retroperitoneal and mesenteric cystic tumors, arising apart from the retroperitoneal organs, although diverse in structure, nevertheless possess a common origin, namely, the wolffian body. The wide structural variations shown by individual cysts are explained by the variations in the structure of different portions of the wolffian body, any one of which may predominate in the cyst. All wolffian cysts have a fibrous wall which frequently contains smooth muscle. The lining consists of columnar or cuboidal, ciliated or non-ciliated epithelium. In the wall may be found, rarely, glomeruli and tubules. The cyst may be multilocular or unilocular, the contents serous, pseudomucinous, chocolate or gelatinous. Maury found chocolate cysts to be significantly frequent. Certain of the unilocular cysts are definitely derived from a persistent wolffian duct. The multilocular cysts are derived from the wolffian body, that is to say, secreting elements. In this latter variety adenomatous and papillomatous proliferation is prone to occur which may lead, as in ovarian cysts, to malignant degeneration. The growth capacity of all forms is great, many cysts reaching a volume of several liters.

Chief among the etiologic factors are age and sex. All cases have been reported in adults with two exceptions (Albarran; Hinman), the patients

being aged 10 and 27 months respectively. Females are affected almost exclusively.

The symptoms produced by wolffian cysts are in no manner characteristic, being due to the mechanical effects of an intra-abdominal mass. To these may be added the effects of malignant invasion of neighboring organs.

The treatment is entirely surgical, which is imposed because of the unlimited growth and the tendency to malignant change.

Since the appearance of Hinman's article (1934) 29 new cases have been published. Of these, 27 have been available to us. The study of these cases bears out in most essentials the conclusions already drawn concerning wolffian cysts as a group. The varieties previously reported reappear together with certain others possessing unusual or significant features. Moreover, with a larger number of cases available it is possible to individualize the wolffian group of cysts somewhat more completely than before.

There are two new examples of cysts containing glomeruli and tubules (Läwen,[10] Sacerdote[23]) which with one of our own brings the total number reported to six. For the first time there appears a wolffian cyst associated with anomalies of the urogenital system (Tscherne[27]), the case of a girl 18 years old having an absent left kidney and ureter and a bicornate uterus.

The most perfect examples of persistent wolffian ducts are those of Läwen, Tscherne, Greig[6] (two cases) and Troup.[26] The latter three were almost identical. They occurred in young children; the ducts led from a sinus at the vaginal orifice upward to the renal fossa. The ducts on removal had the general appearances of a varicose vein. The walls were composed of smooth muscle and fibrous tissue and were lined by transitional epithelium. Two of the patients had been treated for considerable periods for an intractible vaginal discharge. The third presented a vulval swelling.

There were four cysts reported which had developed from the caudal remnant of the wolffian duct (Gärtner's duct). Two of them involved the lateral wall of the uterus near the isthmus.[3, 16] Lordly assigns two vaginal cysts to this category.

Cysts of the hydatid of Morgagni are common but are generally considered to be of little surgical interest. That the latter view is not justified is emphasized by Jeanneney,[9] who reports three cases in which surgical intervention was necessary because of the large size of the cyst or because of complications.

Regarding location, the most unusual was that of Sacerdote, the cyst being attached to the round ligament at the external inguinal ring. The presence of glomeruli and tubules left no doubt as to the origin.

Two characteristics have been stressed which do not appear in the present series, namely, the bloody content of the cysts and the tendency to become malignant. In these cases, when stated, the fluid was serous or pseudomucinous. Only once was it hemorrhagic.[2] In only one instance did the author regard the tumor to be malignant, a tumor of the testis having teratoid features (Monserrat).

Apart from the cases in which glomeruli or ducts were in evidence, the basis for the diagnosis of wolffian cyst was variable.[1, 5, 8, 15, 17, 20, 22, 24] In many, because the cyst was not connected with an organ, the diagnosis was made by exclusion. Much importance has been attached to the presence of ciliated epithelium.[25] Greig, in an exhaustive study, concludes that cilia are not necessarily characteristic of the wolffian body. The presence of smooth muscle in otherwise banal pseudomucinous cysts has been considered to be evidence of a wolffian origin, although these cysts admittedly may be of müllerian origin (Portes). In a cyst of the epididymis and in a cyst of the ovary Ployé and Monnier describe a structure which they regard as specific. They found all transitions from solid cords of epithelial cells to tubules to cysts.

The following personal case is reported because as a wolffian cyst it is rare and is doubly rare in that it combines most of the unusual features thus far observed.

D. W., white, male, two years 2 months old, was admitted to the Arkansas Children's Hospital, October 29, 1934.

Family History.—Father and mother living and well. One sister, a twin, is normal. *Past History.*—The patient was in good health until two months ago. (Four months ago the parents noticed a mass in the left side of the abdomen, a fact learned subsequent to the operation.) The birth and early development were normal.

Present Illness.—The patient entered the hospital with the history of having swallowed a string of wooden beads two months before. Seventeen beads were recovered by an enema but since that time the patient had suffered from diarrhea, 5 to 12 movements daily, and vomiting. There was marked loss of weight. No pus, blood or mucus were observed in the stools. There were no symptoms referable to the urinary tract.

Physical Examination.—A poorly nourished white male child, two years old. Head, negative; ears, nose, eyes, negative; breath sounds, normal; heart, normal; abdomen shows an elevation in the left lower quadrant. Palpation in this region demonstrates a dumb-bell shaped mass which extends upward and laterally somewhat above the level of the umbilicus. The lower half of this mass is the larger and it extends toward the midline; it is firm but elastic, moves with respiration, and its surface is irregular. The left testis is undescended.

Working Diagnosis.—Mass in left lower quadrant with possible partial obstruction of the left colon by remaining beads. Roentgenologic examination of the abdomen was negative. *Blood Count*—Hemoglobin, 70 per cent.; erythrocytes, 5,200,000; while cell count, 16,000; Shilling, 0-1-0-11-12-61-16-7.

Urine Examination.—October 29; clear, trace of albumen, no sugar, occasional squamous epithelial cell, 10 to 15 pus cells per high power field, occasional hyaline cast. November 1, urine clear, straw colored, acid; albumen 2 plus, no sugar, many pus cells, occasional erythrocyte, many bacteria, a few granular casts.

Operation, November 3, 1934: Left rectus incision, appendectomy, removal of undescended testicle. In the left side of the abdomen was a large mass of thin walled cysts almost completely filling the left lower quadrant and extending a little above the level of the umbilicus. The pedicle was attached to the posterior abdominal wall slightly above the level of the umbilicus and to the undescended testicle and its vessels. The colon was pushed toward the midline. The cysts were removed intact. The right kidney appeared normal; the left could not be palpated.

Postoperative Course.—The first four days the temperature ranged from normal to

101.2°. November 9, the dressings were wet with a material resembling urine. Microscopically, the fluid contained much pus. The abdomen at this time became distended. November 10, child began to have convulsions and died in about six hours.

Autopsy: Diagnosis.—Hypostatic hyperemia of the lungs; cloudy swelling of the heart and liver; acute splenic tumor; suppurating laparotomy wound; purulent peritonitis; complete absence of the left kidney and ureter; right hydronephrosis and hydroureter; stricture of the ureter at its junction with the bladder; surgical removal of a left abdominal testis. In detail the situation in the genito-urinary tract was as follows: "The left renal fossa is denuded of peritoneum . . . the spermatic vessels have been sectioned just below the iliac crest. The left vas deferens cannot be discovered. There is no vestige of a left ureter or kidney. The pelvis of the right kidney is markedly dilated and elongated, the vertical measurement being 3.5 cm. The dilated calices can be readily palpated through the thinned parenchyma of the kidney. The right ureter is

FIG. 1.—Urinary tract and cyst photographed in approximately their original relations.

dilated to a diameter of 1 cm. in its upper three-fourths. Below it tapers to enter the bladder. At the point of entrance is a definite constriction which opposes the passage of fluid in either direction. The urinary tract is removed intact." Subsequent gross and microscopic examination of the specimen failed to reveal a spermatic cord or seminal vesicle on the left side.

Pathologic Report.—"The specimen consists of a cluster of thin walled, serous cysts, which form a lobulated oval mass measuring 9 by 7 by 5 cm. (Fig. 1). and a testis. The walls of the cysts are pearly white and they cut with the consistency of dense fibrous tissue. The inner surfaces are smooth and glistening. The cysts number about 15 and they vary from 0.2 to 4.5 cm. in diameter. All obviously arise from a central solid mass of vascular fibrous tissue which measures 3.0 cm. in its greatest diameter.

Microscopic Examination.—The cyst walls are composed of a lining membrane of flat cells, a smooth muscle and a fibrous tunic (Fig. 2). In the central mass and in the thicker portions of the walls of the cysts are numerous well formed glomeruli and tubules (Fig. 3). The tubules are of two types: (1) In close relation to the glomer-

FIG. 2.—Low power (32 obj. 10 × ocular) photomicrograph of the common wall of two small cysts.

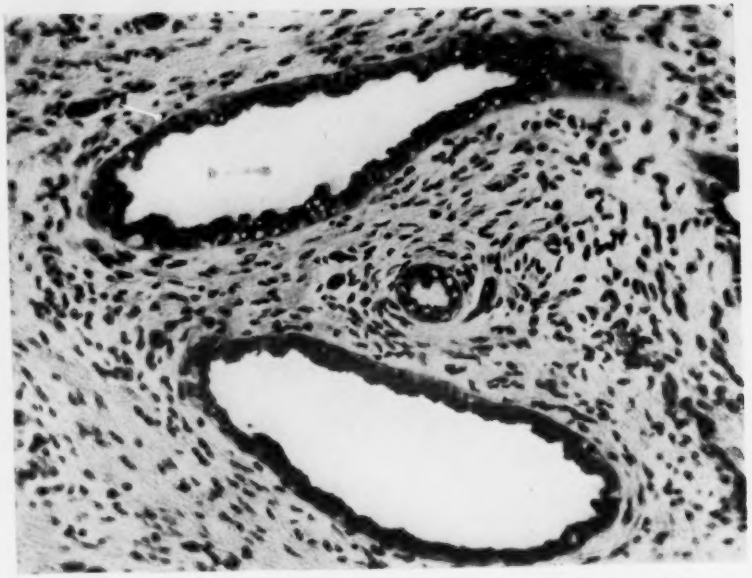
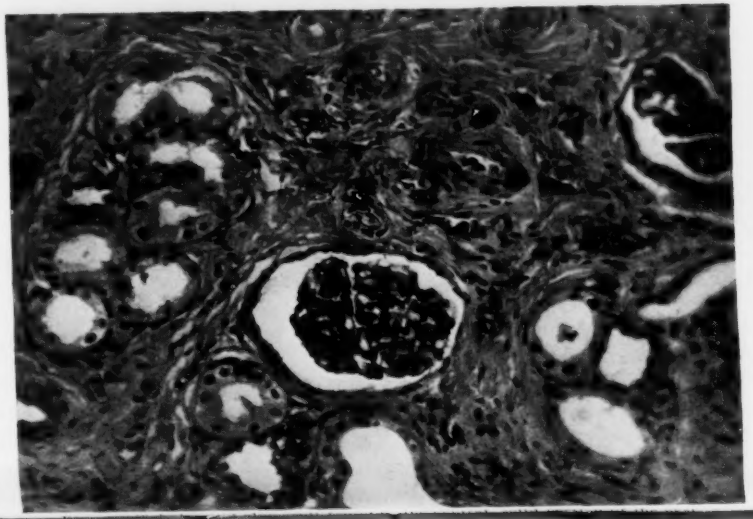
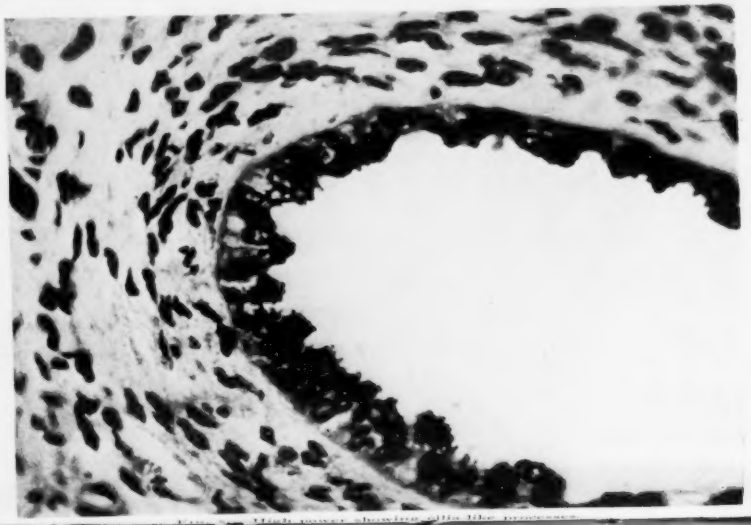


FIG. 4.—Widely distributed type of tubule from which the cysts appeared to develop.





uli are tubules with narrow lumina; the constituent cells are cuboidal, the nuclei are small and vesicular and the cytoplasm is abundant, pale and finely granular. (2) Widely distributed in the cyst walls are tubules formed of cuboidal cells having scanty clear cytoplasm and large, vesicular, deeply staining nuclei. The cells tend to pile up in places and project into the lumina. Some of the cells, particularly at the apices of the projections, possess protoplasmic processes (Figs. 4 and 5). These tubules are found in all stages of cystic dilatation. In none of the above tubules are brush borders observed.

The testis shows the normal infantile structure. The epididymis is not included in the section and the remains of the original block cannot be found.

Diagnosis.—Multilocular serous cyst, probably of wolffian origin. Infantile testis.

Comment.—The cyst could have had one of two origins, the metanephros or the mesonephros. Examination of the embryologic material at our disposal was of no aid in deciding the matter. Histologically, the glomeruli and the tubules immediately associated with them gave every appearance of being derived from the metanephros. It was noted, however, that the larger, dilated tubules in the cyst walls were quite foreign to anything seen in the normal kidney and the presence of cilia-like structures was quite in keeping with a mesonephric origin.

Multiple anomalies of the urogenital tract in association with a wolffian cyst are of interest chiefly because they have been reported only once before. There is no evidence that anomalies play a part in the etiology of cysts of this nature.

DISCUSSION AND CONCLUSIONS

Among the varieties of abdominal tumor which must always be considered in a differential diagnosis is a small group of cysts taking their origin from the wolffian body or duct. These cysts occur almost exclusively in adult females. They may be intramesenteric or retroperitoneal and may be located from the renal fossa to the pelvic floor but are usually found on the left side of the abdomen. They grow slowly and are benign but reach large size. Important attachments to adjacent organs may be acquired which complicate an otherwise simple extirpation. The structure is variable but always includes some component of the wolffian apparatus. A common and characteristic form is a persistent duct which, as Greig has pointed out, is often mistaken for a supernumerary ureter. Equally characteristic but extremely rare are cysts containing glomeruli and tubules. Most commonly reported are unilocular of multilocular cysts which form an apparently heterogeneous group structurally. The diagnosis is usually made on the basis of exclusion or on the basis of certain histologic characteristics which are variously evaluated by different authors. It is only by a careful study of this last group that the actual limits of the category of wolffian cysts will be established.

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RENAL LIPOMATOSIS

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SINCE Kutzmann's scholarly essay on lipomatosis of the kidney, a renewed interest has been taken in this subject. In spite of a rather extensive literature, the pathologic process is little understood, and, like many subjects in medicine, is enmeshed in a fog of theories. However, as case reports accumulate, certain facts are developed which gradually throw light upon the etiology and the manner in which the pathologic changes occur. Many who have studied renal lipomatosis are essentially agreed on several points. These will be taken up separately.

Adipose Tissue in the Renal Parenchyma.—There are two well defined groups exhibiting abnormal adipose invasion of the glandular portions of the kidney. The first is the lipoma. This is usually a well circumscribed encapsuled mass of fat analogous to other growths. It is usually attached to some portion of the renal capsule. As a rule they are not large, but the fat tumor dips well into the parenchyma. In the kidney itself we find no pathology of the glomeruli or tubules, except a very limited area around the adipose mass producing compression. It is possible that Young's recently reported case comes under this group. Examinations of his case failed to show any indications of infection, pyuria or nephrolithiasis. The invasion was from the hilus and involved only a small part of the parenchyma.

In contradistinction, renal lipomatosis gives a picture of renal degeneration, often accompanied by pathologic evidence of infection and then a displacement of the normal glandular structure by fat cells. Tissue specimens from these kidneys often show sharply defined areas where this transformation is taking place. There is enough evidence produced from all the cases reported, except the one by Young,[1] to show that unilateral upper urinary tract infection is a decided factor. All of the microscopic sections show leukocytic invasion and destruction of tubules and glomeruli. In addition, there is noticed occlusion of the renal arteries and veins in the area involved. Some process is at hand which causes this atrophy, either toxic or impaired blood supply. Furthermore, the very high incidence of calculosis in these kidneys has been widely observed and reported. Kutzmann's study of all reported cases had an incidence of 79 per cent of calculus formation.

There is some confusion in the terminology of lipomatosis of the kidney as lipomatous nephritis, replacement lipomatosis (Askanazy), degeneration lipoma (Ulrich), fatty degeneration, lipomatous paranephritis, substitute renes adiposa (Hillman) and other terms. However, authorities are all in accord on the following differentiation. Lipoma of the kidney is a circumscribed fat tumor occurring in normal kidney tissue. In lipomatosis

there are marked atrophic degenerative changes and many are inflammatory with an invasion or substitution of fat cells. Gradually the fat replaces the normal renal infections, thus tuberculous, *B. coli,* and staphylococcus processes can produce a fibrosis of the kidney and bring about a non-functioning condition known as autonephrectomy. These may contain extensive calcium deposits.

Incidence.—The condition is relatively rare. In 40,000 autopsies over a period of 20 years at the Berlin Institution, only one case of fatty tumor of the capsule was encountered. Since Kutzmann's[4] study, in 1931, of 33 cases, 13 new ones have appeared in literature, one each by Kretschmer,[3] Culver[5] and Young, with a joint case report by White and Cambridge.[2] Many of the cases reported were found on exploratory operation or autopsy.

Clinical Symptoms.—There are few outstanding symptoms. Frequently pain is absent. When present it is usually a mild ache and does not radiate. When infection is marked, the usual symptoms of the lower urinary tract are complained of, as frequent micturition and burning.

Objective symptoms are infection of the urine with pyuria. Blood chemistry estimation and renal function tests are often normal, due to the normal kidney on the other side. Roentgenologic studies give a calculus shadow or filling defects similar to renal tumor.

It is a disease chiefly of the fourth or fifth decade of life. The youngest individual reported was ten years of age. It falls within the degenerative period of life. The kidney becomes functionless, hence there is seldom hematuria or colic. It is practically always unilateral. The process is always slow and progressive.

Case Report

Male, aged 77, single, born in Germany, retired optician, was referred by Dr. Harry Friedman July 16, 1930. He was admitted to the Providence Hospital complaining of dysuria. Little of interest was obtained from the family history. *Previous History.*—Childhood infections and bilateral inguinal hernia of 18 years' standing.

The present illness apparently started six years ago. The act of micturition was interfered with by a gradually increasing obstruction producing increased frequency of urination, day and night, a stream of small volume and weakened force, but never a complete obstruction, gradually pain in the pelvis, dysuria, pyuria, mild hematuria and indefinite pain in the small of the back. In time cardiac symptoms developed, *i.e.,* dyspnea after normal body movements, palpitation and a sense of alarm, due to pain over the heart. He was treated for a myocarditis of toxic origin. The dysuria became so distressing that he was hospitalized for a study of his lower urinary tract.

The physical examination showed a small statured, elderly male of good nutrition. The teeth are artificial. The eyes are marked with an arcus senilis. The heart is normal in size but the sounds are weak and distant, somewhat irregular, but no bruit. The abdomen is distinctly globular below the umbilicus, so common in visceroptosis. The right inguinal ring is occupied by a large hernia and the left a smaller one. The kidneys are not

palpable and give no evidence of tenderness. The external genitals are negative. Rectal palpation showed a prostate moderately enlarged, 4 by 5 cm., spherical, with a slightly rough surface. The urine is turbid, blood tinged, specific gravity 1.008, albumin 5.5 per cent by volume, sugar absent, microscopic examination showed many blood cells, numerous pus cells and colon-like bacilli. Culture of the prostatic secretion was rich with the same organism.

Blood examination showed urea nitrogen 15.4 mg. per 100 cc. of blood, creatinin 1.2 mg. and sugar 100.0 mg. Hemoglobin, 55 per cent; erythrocytes, 3,990,000; leukocytes, 10,900; and polymorphonuclears, 70 per cent. Intravenous phthalein returned 25 per cent in the first, and 10 per cent in the second hour.

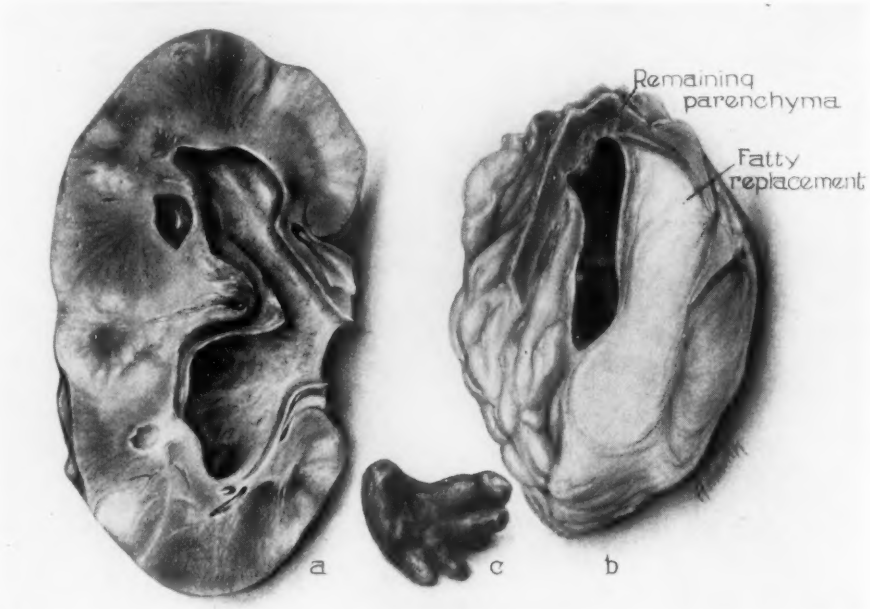

FIG. 1.—Drawing of both kidneys. (a) Right kidney. (b) Left kidney. (c) Calculus found in left kidney.

An indwelling catheter disturbed the patient so much mentally as well as physically that a suprapubic cystostomy was performed July 17, 1930, using novocain infiltration anesthesia. The bladder wall was thickened and its mucosa was greatly inflamed, bathed in hemorrhagic urine. The internal urethral orifice was contracted. A Pezzer catheter was inserted and the bladder drained until August 8. On this date the bladder wound was reopened and the internal urethral orifice was forcibly dilated with the finger. Several pieces of prostatic tissue were removed with scissors. An abscess was located beneath the trigone. The tissue removed was diagnosed as suppurative and inflammatory. Convalescence was slow. In spite of the persisting urinary infection the suprapubic wound healed in three weeks. The patient returned home. Dysuria was not improved so the patient returned to the hospital November 28. A cystoscopic examination was made under spinal anesthesia. The bladder capacity was 90 cc. The urine was purulent. The internal urethral orifice showed edematous tags. The ureteral orifices

were not located due to a diffuse cystitis. The patient very gradually became weaker and died January 2.

Autopsy.—Subject is an emaciated adult white male, appearing about 70 years of age. Suprapubic wound, partially healed, draining purulent fluid.

Body cavities opened in usual manner. Small amount of fluid in both pleural cavities. Right pleura extensively bound by fibrous adhesions, right lung tough and fibrotic. Slight edema of both lungs. Heart is of normal size, but is pale and mottled in color. Depressed area of fibrosis left ventricle. Coronaries narrow with small plaques. Valves competent. Few soft atheromata in aorta.

Abdomen.—Liver is of average size but is pitted by numerous stellate areas as if fibrotic, giving it a nodular appearance and feel. Gallbladder and ducts normal. Liver substance of yellow color. The fatty degeneration stage of chronic passive congestion. Stomach and intestines normal. Spleen is enlarged one and one-half times, with a thick nodular fibrous capsule. Bladder is thick, contracted, and congested. The prostatic area is smooth, urethral orifice patent. Portion of the prostate removed is soft and uniform. Left kidney is intimately bound to perirenal fat and fibrous tissue. On opening only a thin rim of parenchyma is found at one pole, balance has been replaced by fat and fibrous tissue, with a central sac filled with thick creamy pus, also a staghorn calculus (Fig. 1). Right kidney is large, soft and boggy. Capsule stripped with difficulty, revealing an outer surface which is riddled by discrete and confluent yellowish necrotic areas. On longitudinal section yellowish pus was evacuated from the pelvis; parenchyma is shot with necrotic yellowish areas.

Microscopic.—Prostate shows moderate hyperplasia, dense inflammatory infiltration and areas of necrosis. Right kidney.—Discrete and confluent areas of focal inflammatory infiltration, leukocytic and lymphocytic. Considerable necrosis. No evidence of tuberculosis. Degeneration of kidney parenchyma.

Anatomic Diagnosis.—Bilateral pyonephrosis, left renal calculus; chronic cystitis, chronic prostatitis; chronic passive congestion of the liver, numerous healed infarcts; chronic fibrous perisplenitis; chronic myocarditis; edema of lungs; fibrous pleural adhesions, fibrosis right lung.

Tissue Report of the Kidney.—The left kidney measures 10 by 6 by 5 cm. One pole and sides consist of a crescent shaped portion of yellowish-brown tissue, resembling kidney parenchyma. This is sharply marked off from and surrounded by fatty tissue which has replaced all other structures in the kidney. The pelvis and calyces contain a large staghorn calculus measuring 3.5 by 3.5 by 1.5 cm. Portions of the fatty tissue show lobulation.

Microscopic examination of the kidney shows the tissue surrounding the small amount of kidney parenchyma to consist of fatty tissue containing numerous collections of round cells (Fig. 2). There is a very sharp demarcation, separated by fibrous tissue, between the fatty tissue and parenchyma (Fig. 3).

The parenchyma shows an extensive destruction and fibrosis of the glomeruli and tubules with areas of hyaline degeneration, together with a diffuse infiltration and collections of round cells (Fig. 4). The smaller arteries show narrowed lumen, marked thickening of the wall by fibrosis and hyaline changes.

Pathologic Diagnosis.—Replacement lipomatosis of the left kidney; pyonephrosis; renal calculus.

FIG. 2.—Photomicrograph (low power) showing the structure of adipose tissue which has replaced the kidney parenchyma. Note the diffuse round cell infiltration.

FIG. 3.—Photomicrograph (low power) showing the sharp demarcation between the adipose tissue and the fibrous renal parenchyma.

The interesting lessons taught by this particular case are, first, that chronic infection of the lower urinary tract in the aged usually involves the kidneys; second, that the blood chemistry estimation is often misleading as to the true extent of renal damage by infection, and third, when lower urinary tract pathology exists a complete roentgenologic study should be made of the entire urinary system whenever possible.

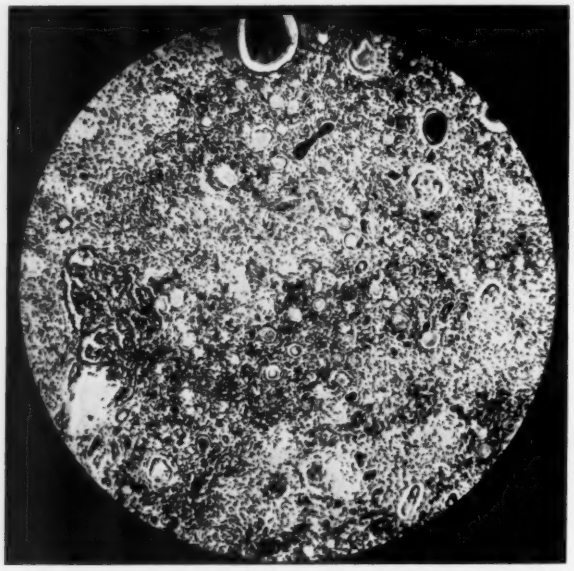

FIG. 4.—Photomicrograph (low power) showing the remaining renal parenchyma, with destroyed fibrous areas, marked inflammatory cellular infiltration. Many miliary abscesses scattered throughout.

SUMMARY

(1) Renal lipomatosis is the result of a degenerative atrophic process, due to bacterial toxins. Renal lipoma shows no degenerative changes in the parenchyma, thus differentiating it from replacement lipomatosis.

(2) The normal kidney cells become altered. Globules of fat replace the contents of the cells.

(3) The action of bacterial toxins and lack of oxygen bring about a cell degeneration.

(4) The degeneration is primary and the fat replacement secondary.

(5) Nephrolithiasis almost invariably is a result of these changes.

(6) Symptoms and diagnosis are often obscure. Most cases are found on exploration or autopsy.

(7) Lipomatosis is a disease of the degenerative period of life.

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TRUE HERMAPHRODITISM

REPORT OF A CASE WITH NECROPSY

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THE medical literature contains many references to hermaphroditism. Despite the widespread interest in this condition, the confusing terminology and the paucity of pertinent data in many reported cases make this one of the least understood subjects in medicine.

True physiologic hermaphroditism would necessitate functioning glands and accessory organs of both sexes in one person. The nearest approach to this condition was a case with bilateral ovaries and testes reported by Masson,[1] who found menstrual blood in the uterus and spermatozoa in the ducts. Kwartin and Hyams[2] were unable to find a higher stage of spermatogenesis than spermatocytes in the photomicrographs from Masson's patient and it is generally conceded that true physiologic hermaphroditism does not occur in vertebrates.

The term true or glandular hermaphroditism is applied correctly only to individuals with both male and female gonads. The glands may be discrete or fused to form an ovotestis. Broman[3] divided true anatomic hermaphroditism into three groups:

(I) Hermaphroditismus bilateralis. Testis and ovary present on both sides (either separate or united ovotestis).

(II) Hermaphroditismus unilateralis. On one side testis and ovary present. On the other side either one or the other.

(III) Hermaphroditismus alternans. Testis on one side and ovary on the opposite side. (The alternans also called lateralis.)

Kwartin and Hyams[2] reported a case in 1927 and found 11 other proven cases in a critical review of the literature. They conclude: there is always a mixture of external male and female sexual characteristics; most undoubted cases possess either unilateral or bilateral ovotestis; the ovarian part of the ovotestis usually lies cephalad, the testicular dorso-caudad; the testicular component is usually larger in size than the ovarian; the ovarian portion is almost always highly differentiated, with activity on the part of the ovarian stroma; the testicular portion is small, atrophic or rudimentary and histologically exhibits more or less completely the features of a cryptorchid testis without mature spermatozoa although the interstitial cells may be well developed, especially after puberty.

Kwartin and Hyam's[2] collected series of true hermaphroditism included only three cases occurring in America (Masson,[1] Young[4] and their own case). Essenberg and Feinberg[5] have since reported a microscopically proven

case. McFarland[6] and Urechia and Teposu[7] have recently reported undoubted cases of true hermaphroditism although they were able to obtain microscopic sections from only one type of gonad.

The term pseudo or tubular hermaphroditism has been loosely applied to many conditions. Strictly speaking, this term includes any persistence of the müllerian or wolffian ducts beyond the vestigial remains usually found in normal persons. These embryonic remains may vary in differentiation from a simple tubular structure accidentally encountered at operation or necropsy to the completely differentiated tubular accessory structures of the opposite sex. Heyl's[8] recent article reporting a case of probably minimal pseudohermaphroditism found during the repair of an inguinal hernia suggests that this condition may frequently pass unrecognized. Highly differentiated pseudohermaphrodites can be distinguished from true hermaphodites only by careful microscopic examination of the gonads, and inability or failure to do this will leave the status of a case undetermined.

The interesting question of the relation of hermaphroditism to sex differentiation is not discussed in this paper. The significance of this relationship has been presented admirably by Jordan[9] and Jordan and Kindred.[10]

Case Report

H. M., an elderly Negro, who appeared older than the stated age of 60, was admitted to Saint Philip Hospital October 31, 1933, complaining of abdominal pain and vomiting of four days' duration.

The family history was not significant. Little information could be obtained regarding the health or cause of death of the patient's immediate relatives. No congenital abnormalities were recalled.

The past history was fragmentary and was obtained from a relative following the death of the patient. A large right inguinal hernia had been present since birth and had handicapped the patient to such a degree that heavy manual labor had been impossible. The presence of the hernia and a marked hypospadias was known to the patient's associates during childhood and had given rise to the supposition that the subject was a hermaphrodite. This early impression apparently was refuted when upon reaching maturity the patient married. The union proved mutually satisfactory and was terminated only by the death of the wife from tuberculosis. This was followed by a second marriage which ended in a divorce when the wife proved unfaithful during the patient's absence from the city. A third marriage in middle life was followed by a separation because of conflicting personalities. No pregnancies resulted from these unions. The third wife stated that the patient was not impotent. A history of periodic hematuria could not be obtained. In recent years the patient's activities were limited by the inguinal hernia. About six months before admission to the hospital weakness and loss of weight were noted, but the patient attributed this to advancing years and the ever increasing hernia, which was then irreducible.

The present illness was of short duration. On October 27, the hernia became larger, the patient developed cramping abdominal pain and vomited several times. These symptoms persisted and the patient was admitted to the hospital four days after the onset of the obstruction.

Physical examination revealed an acutely ill, elderly individual, apparently masculine, of medium statue. Although the patient had a moderate beard, the breasts were rather large, the hips were wide and the thighs were definitely feminine in contour. The head and neck were negative aside from advanced dental caries. The

heart was enlarged, the apical impulse was situated in the sixth interspace 4 cm. lateral to the midclavicular line. The blood pressure was 140/85. The abdomen was distended and generally tender. A fulness in the region of the right inguinal canal was continuous with an irreducible right inguinal hernia which measured 69 cm. in circumference (Fig. 1). The scrotum was tense and the skin was ulcerated where the hernial protrusion rested upon the inner surface of the thighs. The penis was hidden in a sulcus in the upper medial portion of the hernia. Temperature, 99°; pulse, 120;

FIG. 1.—Preoperative photograph showing size of hernia. Note distribution of pubic hair and contour of pelvis and thighs. The penis is almost completely hidden by the hernial protrusion.

and respirations, 24. The patient was catheterized with difficulty due to the hypospadias and invagination of the penis in the scrotum. Urinalysis negative except for a slight trace of albumin. Routine blood studies were not remarkable. The patient obviously had an intestinal obstruction resulting probably from a strangulated right inguinal hernia. A continuous intravenous injection of 5 per cent dextrose in normal saline solution was begun. A gastric lavage released a large amount of gas but no gastric contents.

Operation.—Twelve milligrams of pantocain were injected intraspinally. The hernial sac contained a large amount of omentum, cecum, ascending colon, and the greater

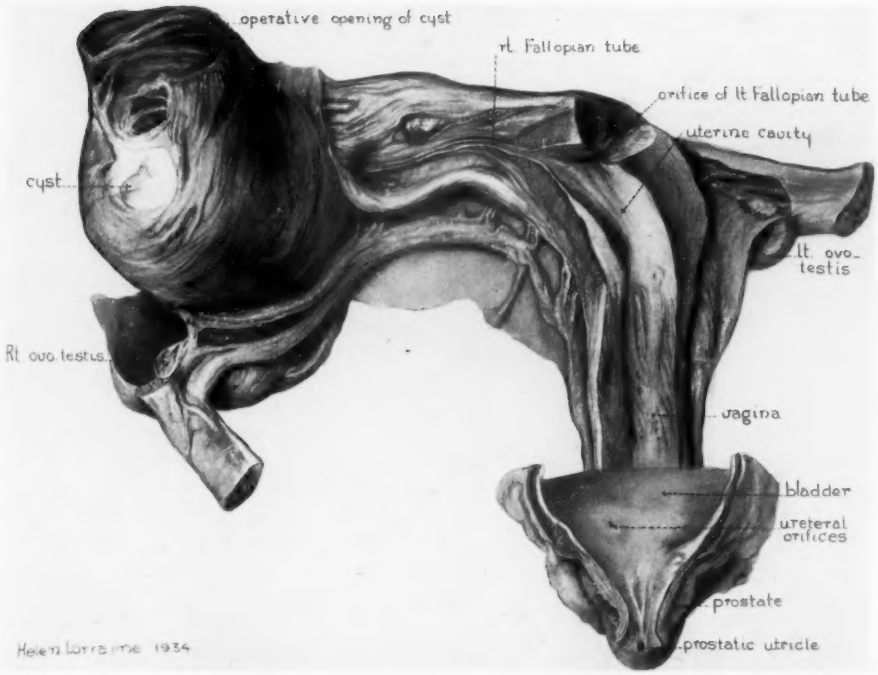

FIG. 2.—Drawing showing anterior view of specimen.

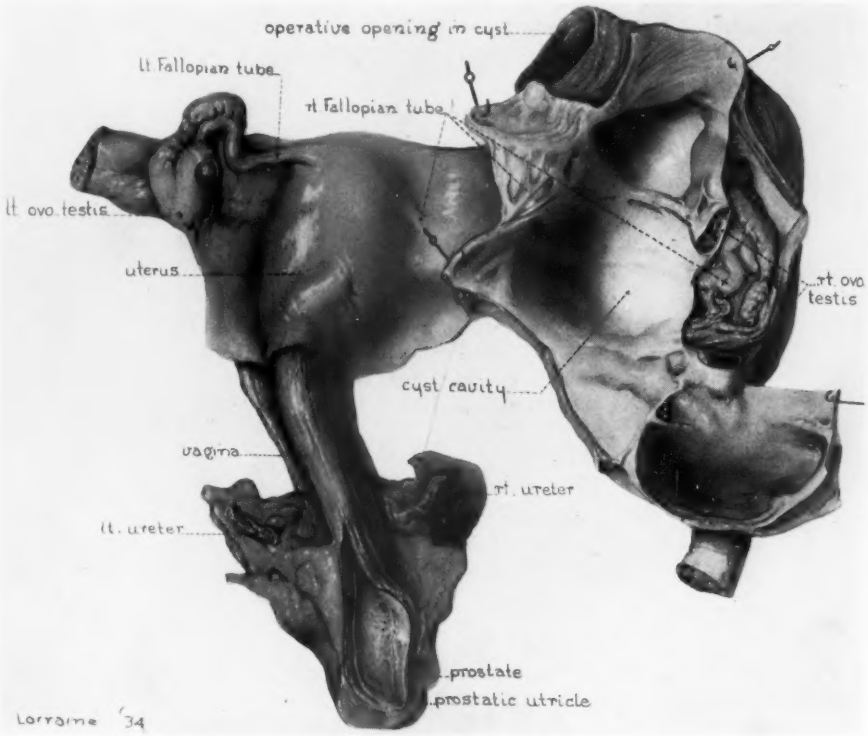

FIG. 3.—Drawing showing posterior view of specimen.

portion of the ileum. The serosa of the omentum and bowel was studded with small gray elevated nodules which resembled peritoneal implants from an intra-abdominal carcinoma. The primary obstruction was located in the terminal ileum where adherent metastatic nodules bound the adjacent loops of intestine together with angulation and a complete obstruction at this point. The resulting dilatation of the ileum proximal to the angulation had in turn caused a secondary obstruction at the neck of the sac with marked distention of the jejunum. A biopsy from the omentum showed metastatic adenocarcinoma probably arising from the pancreas. In dissecting free the adherent loops of small intestine a cyst about 10 cm. in diameter was found adherent to the posterior wall of the hernial sac. This contained straw colored fluid and was thought to be a hydrocele. Adherent to and projecting from the outer wall of the cyst was a small solid structure which resembled a cryptorchid testis. These structures were continuous with a large unidentified organ within the abdominal cavity. The condition of the patient was unsatisfactory and a more detailed examination could not be made. The obstruction was released, the contents of the sac were returned with difficulty to the abdominal cavity and the hernia was repaired. In view of the marked distention a catheter jejunostomy was made through a left muscle splitting incision.

The patient improved for a short time following operation but the abdomen again became distended, the non-protein nitrogen of the blood increased, the extensive ulceration on the under surface of the scrotum bled freely and death occurred three days after operation.

Necropsy.—(Abstracted to include only relevant data.) Gross description: Case A 1139. Body examined November 3, 1933, five hours after death. Well nourished Negro. Apparent age, 65 years; length, 160 cm.; estimated weight, 65 Kg. The beard and mustache are developed to a moderate degree. The mammary glands are considerably larger than usually is observed in males, and the nipples are quite prominent. The width and contour of the pelvis and the conformation of the lower extremities are very suggestive of the female type. The pubic region has been shaved for surgical procedures. The scrotum, which is excoriated superficially, measures approximately 20 cm. in diameter, and almost obscures the short penis which presents a penoscrotal hypospadias. The testes are not contained in the scrotum.

Median abdominal section reveals the following anomalous formations: Projecting upward between the urinary bladder and the rectum is an unusual bulky muscular structure which resembles a malformed uterus (Figs. 2 and 3). The upper portion of this organ is loosely attached to the lateral walls of the pelvis by relaxed ligamentous structures. The lower portion is adherent to the prostatic urethra near the base of the bladder. It contains a central canal which communicates with the prostatic urethra through the utricle orifice, the latter being located in the usual position. A probe passes freely through this orifice and enters the canal which measures 8 cm. in length and about 1 cm. in diameter. This portion suggests a vagina, and is lined with a glistening mucosa which presents longitudinal rugae. The cervix is not well defined. The upper end of this vaginal-like structure merges into a thick walled portion resembling the fundus uteri. The uterine cavity is 10 cm. in depth and the wall varies from 1.5 to 2 cm. in thickness. It is grossly identical with myometrium. The fundus deviates to the right and extends into the right cornu, where it diminishes markedly in caliber and emerges on the posterior surface of the right ligamentous structure as the fallopian tube.

This tube is 12 cm. in length and tortuous near the end of its course; it terminates abruptly without a fimbriated extremity near a firm ovoid-shaped structure. This mass measures 2.5 by 1 by 1 cm. in size and is grayish-red in color. Section reveals a firm reddish and mottled gray surface containing several cystic spaces. Medial and slightly posteriorly is situated a smooth walled evacuated cyst which measures approximately 10 cm. in diameter.

The left fallopian tube arises from the somewhat ill defined left cornu of the

uterus. This tube measures 4.5 cm. in length and terminates in an ovoid shaped firm mass which is 2.75 by 1.5 by 1.25 cm. in size. It is pale, reddish-gray in color. Section reveals a small cyst associated with calcified material. The remainder of the surface is rather homogeneous and dark red. Superimposed upon this ovoid mass and in-

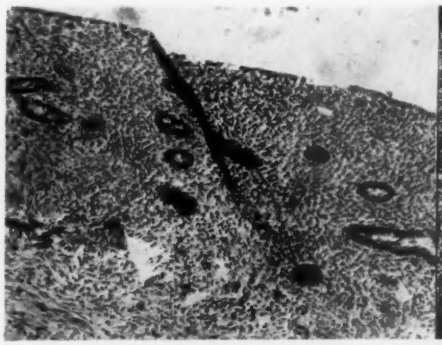

FIG. 4.—Section showing atrophic endometrium and a portion of the myometrium.

FIG. 5.—Section from left ovotestis showing both testicular and ovarian elements.

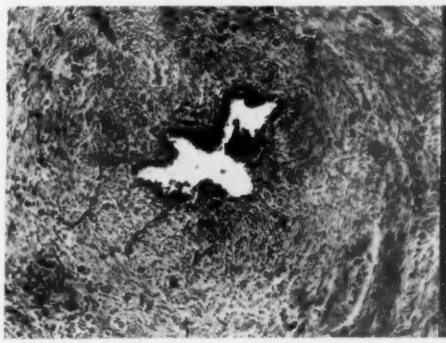

FIG. 6.—Cross section of left fallopian tube near uterine end.

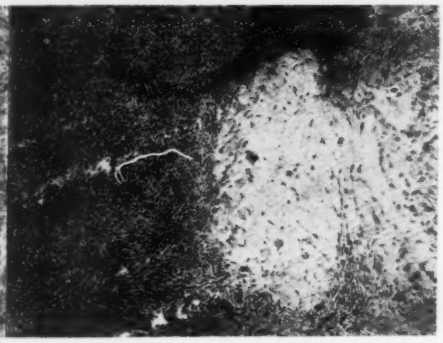

FIG. 7.—Section from right ovotestis, showing ovarian stroma and a corpus albicans.

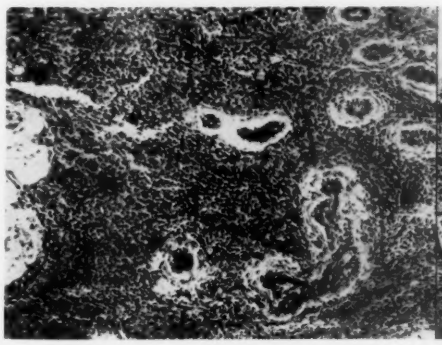

FIG. 8.—Another view of right ovotestis, showing mainly testicular elements. From near opposite end of organ seen in Fig. 7.

FIG. 9.—Cross section of right fallopian tube near the distal extremity.

separably associated with it is a grayish-white convoluted structure of delicate configuration. Fimbriated extremity cannot be demonstrated.

A small shallow prostate surrounds the urethra in the usual location. Seminal vesicles and vasa deferentia cannot be demonstrated grossly.

Small metastatic carcinomatous nodules are sparsely distributed in various areas of these pelvic structures. The carcinoma is primary in the pancreas.

Microscopic Examination.—The fusiform muscular structure reveals myometrium and atrophic endometrium (Fig. 4).

Section from the ovoid shaped mass on the left side shows ovarian stroma embracing corpora albicantia (Fig. 5). Testicular tissue is noted in another portion of the section which is continuous with the ovarian elements. The ovarian elements assume a cortical location while the testicular tissue is centrally placed or medullary. The convoluted structure noted in the gross associated with this ovotestis shows the configuration of an epididymis.

Section of the left tubal structure from near the uterine end is identified as fallopian tube from that location (Fig. 6).

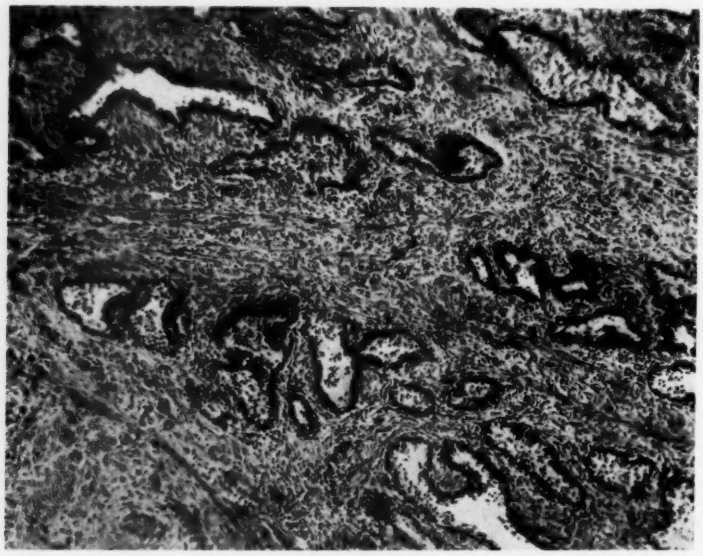

FIG. 10.—Section from prostate gland.

The ovoid shaped mass on the right side presents a somewhat different position of predominating elements. Section selected from nearer one end presents mainly ovarian stroma and corpora albicantia (Fig. 7) associated with an inconspicuous number of cryptorchid testicular elements embracing atrophic seminiferous tubules which are hyalinizing and disappearing. In sections removed from nearer the opposite end the elements are predominantly testicular in character (Fig. 8), the interstitial cells being very abundant. This observation of distribution of various elements was made by Doctor Jordan, who also identified the region of transition from epididymis to ductus deferens in another section which was removed from close proximity to this right ovotestis.

Section from near the termination of the thin walled narrow channel, or continuation of the uterine canal towards the right, presents, the histologic picture of fallopian tube (Fig. 9).

The prostate (Fig. 10) presents a typical picture of the glandular and stroma elements of that organ. The appearance is compatible with prostatic tissue of a much younger individual than the subject of this report.

Breast section shows an occasional lactiferous duct and a few atrophic acini.

SUMMARY

(1) A case of true anatomic bilateral hermaphroditism in an elderly Negro is reported. The following structures were identified: bilateral ovotestes; fallopian tubes; a uterus with a distal prolongation which grossly resembled a vagina; epididymi; and a prostate. A ductus deferens was found on one side. The penis presented a penoscrotal hypospadias.

(2) This anomaly was associated with a large congenital inguinal hernia containing a cyst which arose near the right ovotestis.

(3) The patient considered himself male. He married three times and apparently led a normal sex life.

(4) The incidence and various types of true hermaphroditism are given but no attempt has been made to discuss the theories as to the causation of this condition.

We are indebted to Dr. H. E. Jordan, Professor of Embryology and Histology at the University of Virginia, School of Medicine, and to Dr. Hjalmar Osterud, Professor of Anatomy, at the Medical College of Virginia, for assistance in this study.

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PRIMARY CARCINOMA OF THE EXTREMITIES

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THIS report is part of a general study of the clinical aspects of soft tissue tumors of the extremities. The intention has been to emphasize the clinical syndrome presented by each of these tumors in order to facilitate early diagnosis and better prognosis. Even though their pathology has been adequately described, a high percentage of false preoperative diagnoses has heretofore marked every series of hospital records examined. This has been due entirely to a failure to appreciate their clinical differential characteristics. Those of carcinoma are herewith presented.

Primary carcinoma occurs in the extremities infrequently but by no means rarely. In 1926, De Asis collected 17 cases[1]; in Stout's series of 51 skin epitheliomata, 17 involved the extremities.[2] The present study is based upon 21 cases collected from hospital records, where sufficient clinical data warranted consideration. The relative frequency of primary carcinoma as compared to other soft tissue tumors of the extremities is shown in Table I.

TABLE I

Lipoma	54%
Fibroma (including so called fibrolipoma)	12%
Haemangioma	10%
Tumors of nerve sheath origin (neurofibrosarcoma)	8%
Fibrosarcoma	8%
Primary carcinoma	5%
Xanthoma	3%

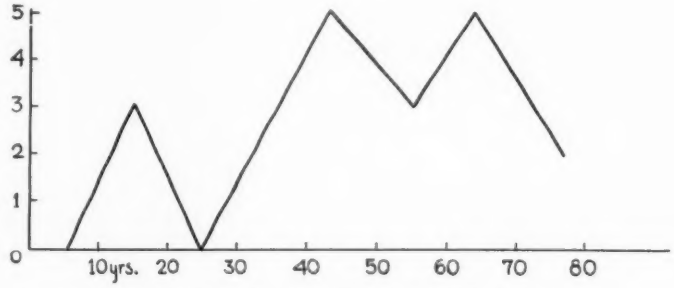
Showing percentages of occurrence of various types of tumors in extremities in a series of 300 cases.

Of the many histologic elements which go to make up a human limb, only the skin can be the point of origin for primary carcinoma. The other tissues are all of mesodermal or endodermal origin, with the exception, of course, of the nerve fiber, but this is a cellular process, or axone, whose cellular elements lie within or adjacent to the spinal cord. Hence, primary carcinomata of the extremities are epitheliomata. Three types are distinguished: squamous cell, basal cell, and melanotic carcinoma. The first is derived from the cellular stroma of the dermis; the second, which includes the rodent ulcer type, from the basal layer cells of the corium, hair follicles, sweat and sebaceous glands; the third, from pigment containing cells. Each of these types possesses clinical as well as pathologic characteristics. The last of these, melanocarcinomata, are considered rare. This may be true in reference to the body as a whole, since epitheliomata of all types are far more common about the face and neck

than on the extremities. However, in our series two were melanotic, so that, until further case groups are collected, we must assume that in primary carcinoma of the extremities about 10 per cent may be melanotic. This is of utmost importance since of the three types the melanotic is the only one which possesses a high grade of malignancy. Of the remaining cases, six were classified as basal cell, four as squamous cell, and nine were unclassified other than being epitheliomata. The frequent inability to differentiate the two types of nonpigmented epitheliomata was also noted by De Asis.

Sex.—Eleven cases occurred in males and ten in females. In previous reports, males were said to predominate (Broders,[3] four to one, De Asis, 12 to five). In view of our own series, it appears that this predominance may be far less marked.

Etiology.—In no other soft tissue tumor of the extremity is the relationship between trauma and tumor so evident as in primary carcinoma. This trauma may be direct, following a crushing injury; or indirect, for example,



GRAPH 1.—Showing age group frequency.

developing within the scar of an old burn or other irritative lesion. Ullman[4] noted that 80 per cent of skin carcinomata found associated with scars occurred in the scars of burns. In our series this figure was not even approached, only two of five scar epitheliomata having followed burns (40 per cent). Infection occurring in old operative scars was of equal etiologic importance.

The association of these neoplasms with ulcers must be noted. In 12 of the cases ulceration was present. In some the ulcer was of such long duration and so obviously the result of infection or trauma that it could with reasonable certainty be considered primary and the carcinoma secondary to it. In other of the cases, however, the tumor was probably primary in an infected sinus or unepithelialized portion of a scar, and subsequently became itself ulcerated. In at least three of the cases the ulcer represented the tumor as an uncompleted phenomenon forming a fungating mass. To differentiate these instances is speculative and of academic interest only. Suffice it to emphasize the frequency of association between primary carcinoma of the extremities and ulcer. In none of the cases was the ulcer of varicose origin.

Age of Onset.—This ranged from 13 to 73 years (Graph 1). The three cases which occurred under 20 years of age each presented unusual features. Case I was considered a naevocarcinoma of the pigmented variety,

i.e., melanocarcinoma. Case VIII was a squamous cell carcinoma occurring in the stump of a limb amputated following the prolonged treatment of an infected scar from a severe burn sustained in early childhood. Case XIII was diagnosed as an epithelioma (unclassified), excised following ineffective radiation. Excluding these three cases the others fell into the cancer age span, over 30 years.

Duration.—The average duration of original tumors, where this could be clearly determined, was 3.2 years. However, in many instances, the time of onset could not be determined because of a complex history of recurrent ulceration in a scar or wound.

Location.—Because of its association with scars, ulcers and trauma noted above, there is no usual site of predilection for primary carcinoma as there is for most of the other common soft tissue tumors of the extremities. Of the four cases involving the upper extremity, two occurred on the forearm, one on the hand, and one on a finger. Of the 17 cases involving the lower extremity, six occurred on the thigh, four each on the foot and leg, and three on the toes. The preponderance of lower extremity cases merely marks the greater frequency of trauma and infection on the lower as compared to the upper extremity.

Pain.—An important diagnostic characteristic of these tumors is the occurrence of pain, which often is the occasion for renewed interest in an old "weeping" scar or neglected sore. We have pointed out elsewhere that among the soft tissue sarcomata of the extremities pain is rarely a prominent symptom.[5, 6] Only in the active stage of neurofibrosarcoma is the pain comparable in severity to that of carcinoma.[7] It should become a surgical axiom that the appearance of pain in an ulcer or sinus after a period of apparent quiescence marks the onset of malignancy until proven otherwise by careful biopsy.

Physical Characteristics.—When the tumor presents as a palpable nodule, it is of stony hard consistency. However, as noted above, it more often appears as a diffuse, amorphous mass, frequently fungating or ulcerous, and then its consistency is indistinguishable from the host tissue. Its size varies from that of a pea to a grapefruit, but ordinarily attains the diameter of a half dollar, or the size of a walnut.

Metastasis.—Of the malignant soft tissue tumors of the extremities, carcinoma is the only one which metastasizes by way of the regional lymphatics[8] (there are a few very rare exceptions to this). Hence, to all practical purposes, a palpable inguinal or axillary node in the presence of a soft tissue tumor of the extremity is pathognomonic of carcinoma. For the most part primary carcinoma of an extremity is of low grade malignancy and does not metastasize rapidly. The average duration of the lesion before admission (3.2 years), attests to this fact. In many instances metastases occurred only after a long period of hesitant and ineffective treatment.

Recurrence.—In five of the present series (24 per cent), a definite history of recurrence was established. This figure is subject to considerable theoretical modification. In several additional cases it could not be definitely de-

TABLE II

ANALYSIS OF 21 CASES OF PRIMARY CARCINOMA OF THE EXTREMITIES

	Diagnosis	Location	Sex	Age	Duration	Size	Pain	Palpation	Tissue of Origin	Treatment	Remarks
1.	Carcinoma, probably nævo	Outer, lower ⅔ Thigh	M.	20	7 yrs.	Grapefruit	No	Hard mass	Deep structures of corium	Excision and roentgen ray	Thought at first to be fibrosarcoma; recurrence 1½ yrs. later with metastasis to lymph nodes, proved to be carcinoma
2.	Carcinoma, basal cell	In scar of burn sustained in childhood	F.	55	Original tumor 2 yrs. Recurrence 7 mos.	Plum	No	Fluctuant (necrotic)	Scar (burn)	Original roentgen ray followed by excision. Recurrence excised	Recurrence; ulcerated
3.	Melanocarcinoma.	Big toe	M.	55	7 yrs.		Yes		Crushed tissue —amputation stump		Toe had been amputated 7 yrs. after crushing injury; followed by radium without relief. Ulcerated 3 yrs. after amputation (present admission) metastases were found in groin
4.	Carcinoma.....	Anterior, upper ⅓ leg	F.	35	4 mos.	Pea	No		Skin	Radium	Ulcerated
5.	Carcinoma, basal cell	Upper ⅓ thigh	M.	68	1.5 yrs.	Plum	No	Hard	Skin	Radium and excision	Ulcerated; regional nodes involved
6.	Carcinoma, squamous cell	Anterior surface, knee	M.	73	2 yrs.	Grape	Yes	Hard	Skin	Radiation. Oper. refused	In scar of old infected operative wound; ulcerated—fungating mass
7.	Carcinoma.....	Anterior, middle ⅓ leg	M.	67	14 yrs.	Plum	Yes		Skin	Excised	In scar of wound sustained at age of 20 ulcerated. Excised at 53—healed. Recurred
8.	Carcinoma, squamous cell	Thigh, amputation stump	M.	20	1 yr.				Scar of stump	Excision — reamputation	Burned knee at 6 yrs. Infected —ulcerated. Amputated at 19 yrs. Ulcer reappeared
9.	Carcinoma, basal cell	Inner, lower ⅓ thigh	F.	52	3 yrs.	Plum			Skin	Excised	Ulcerated
10.	Carcinoma, squamous cell	Heel	F.	63	1 yr.				Skin	Excised	Started as "pimple"; infected; ulcerated
11.	Carcinoma.....	Thigh	F.	54					Skin	Excision radiation	Below groin
12.	Carcinoma.....	Plantar surface foot	M.	22	3 yrs.		Yes	Hard	Skin	Excision radiation	Radiation ineffective
13.	Carcinoma.....	Forearm, anterior	M.	70	10 d. (?)		Yes	Hard	Skin		Refused treatment
14.	Carcinoma, squamous cell	Legs, bilateral anterior surfaces	M.	67	20 yrs.				Skin	Excision radiation	Recurrent ulcers
15.	Carcinoma, basal cell	Toe	F.	39	1 yr.	Grape	Yes		Skin	Excision	
16.	Carcinoma, basal cell	Finger	F.	65	2 yrs.				Skin	Excised	
17.	Carcinoma.....	Heel	M.	49	8 yrs.	½ dollar			Skin	Excised	Ulcerated
18.	Carcinoma, basal cell	Foot, plantar surface	F.	77	4 yrs.	Walnut	Yes		Skin	Radiation 10 mos. ago	One wk. before admission; metastases noted at groin
19.	Carcinoma.....	Upper, inner thigh	F.	60	2 yrs.				Skin		Ulcerated; had previously been treated by roentgen ray and radium
20.	Carcinoma.....	Hand	F.	70	9 mos.				Skin	Amputation	Site of congenital contracture of index finger; ulcerated
21.	Carcinoma, squamous cell	Toe	M.	38	1 mo. (?)				Skin	Elect. coag.	Developed after using corn plaster for several months

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termined whether a recurrence had occurred or merely a continuation of an insufficiently treated original lesion. Since probability in these cases favored the latter belief, they were not included among the recurrences. On the other hand, several of the recurrent cases had been treated with radium or roentgen ray and declared cured at the time. This was of course an arbitrary assumption. Since both of these exceptions are statistically mutually antagonistic, the established figure for recurrence, 24 per cent, may be assumed to come within a reasonable margin of error (Table II).

Treatment.—This has been for the most part unsatisfactory. Too often in the history of these cases there was noted long and varied local treatments in the form of medical applications or cauterizing measures to ulcerating tissues. This, it must be assumed, was due to false diagnosis, and could have been avoided by biopsy. While radiation has been the treatment of choice in most clinics, and, with recent improvement in technic,[9] will no doubt become increasingly efficient, it has been far from uniformly successful. In six cases, including basal and squamous cell tumors, where radium or deep roentgen ray therapy had been used, the progress of the neoplasm was not averted. On the basis of present observations, the following procedures must be considered advisable.

(1) In primary carcinoma involving the digits, amputation of the digit should be performed without hesitation, followed by radiation of the adjacent tissue and regional lymph nodes.

(2) In primary carcinoma of other parts of the extremities, either wide excision of the tumor including adjacent host tissue, or amputation, followed by properly conducted radiation, should be instituted, including the local area and regional nodes. Whether wide excision or amputation is chosen depends entirely upon the grade of malignancy of the tumor. This must be determined by the history of the case plus biopsy, rather than biopsy alone, since basal cell carcinoma frequently presents a microscopic appearance of indolence in spite of obvious rapid growth clinically.

(3) Melanocarcinoma necessitates immediate amputation and radiation of the regional lymph nodes, but with a guarded prognosis at best.

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ELECTROSURGERY IN ADVANCED CARCINOMA *

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MANY cancer patients who come for treatment are in such advanced stages that they are pronounced inoperable by the ordinary surgical methods. Should roentgen ray and radium therapy fail to arrest the disease, they are usually considered to be beyond all hope of aid. However, among these patients there are many for whom electrosurgery may still furnish good, and frequently lasting, palliative results of even five years' arrest. Keysser,[1] in 1933, at the Cancer Congress in Madrid, reported 17.2 per cent of five year cures.

Electrosurgery, in our experience, appears to be the most effective method of treating ulcerated, secondarily infected carcinomata of the body surface and cavities. The experiences of Clark, Wyeth,[2] Keysser,[3] Seeman,[4] Ward[5] and others have established the efficacy of electrical procedures in the fight against cancer. Among its special advantages is the minimal blood loss during operation, an especially important factor in the patient weakened by cancer. Moreover, the destruction of infected areas by carbonization and their fixation by coagulation minimizes the likelihood of generalized infection. Another advantage is the rendering sterile of the operated area for cancerous cells and microorganisms, for which reason a plastic operation may be done with greater safety at the same time. The operative dissemination of tumor cells is reduced because the electric cutting process destroys the cancer cells.

During the operation it is possible to recognize and to follow the cancerous tissues wherever they extend grossly, because the coagulated tumor tissues present a different color and consistency easily recognized by the experienced operator. Hence, the macroscopic extent of tumor may be determined with greater certitude than when operation is done with the scalpel. If the wound is left open the coagulated tissues form a protective layer over the deeper tissues against outside irritations and bacterial invasion, provided a sterile technic is employed in the after care. This facilitates multiple stage operations if the extent of the tumor or the patient's condition forbids a one stage procedure.

Electrosurgery and irradiation therapy in combination often afford a more favorable result than either method alone. Following the removal of a bulky infected tumor a more effective use of the resources of radiation is made possible.

Our experiments on rats with sarcomata and Flexner carcinomata seem to indicate that coagulation of these tumors, even if in sudden ebullition the tissues explode, does not cause distant metastasis any more frequently than

* Read before the New York Surgical Society, March 27, 1935.

occurs in control animals. Furthermore, we could not experimentally affirm the clinical observation that the absorption of coagulated tumor tissue favorably influences the regression of other tumor rests. However, in spite of the fact that the latter hope does not seem to be fulfilled by electrosurgery, the method has certain undeniable advantages. We have used this procedure on a large number of cases in the Skin and Cancer Unit of the New York Post-Graduate Hospital with only 3 per cent immediate postoperative mortality.

Although the operation was extensive in many instances there was no postoperative shock. Moreover, during convalescence there was relatively so little pain that many of the patients did not require narcotics.

Electrosurgery is not a shocking procedure and is well borne even by the cachectic patient. It is, therefore, a most effective palliative measure to prolong life and give comfort even to patients presenting distant metastases.

Four cases treated by electrosurgery alone are presented. They are representative of four types of procedure and demonstrate that electrosurgery can still give relief in cases which, by ordinary criteria, would seem to be hopeless.

Illustrative Case Reports

Case I.—Advanced squamous cell carcinoma of the tongue (No. 31–2247. Path. No. 15,755). This patient, a white male, 79 years of age, came to Stuyvesant Square Hospital presenting an extensive hard mass on the left side of his tongue measuring 3 by 2 cm. which he had noticed about four months previously (Fig. 1). The pathologic report was squamous cell carcinoma.

A year and a half before the patient came to us, he had been treated by implantation of radium needles. Although a total dose of 968 mgh. was followed by slight improvement, the tumor persisted, increased in size, and became painful. On examination the patient was in poor condition. Red blood cell count, 1,600,000. There was leukoplakia over the anterior third and an ulcerated, firm area about one inch in diameter at the left side of the tongue, the lesion extending towards the midline and towards the base of the tongue. No submaxillary nodes were palpable.

Under local novocain anesthesia, the lesion was removed electrosurgically, following the tumor step by step, with a minimum loss of blood. The freshened wound edges were approximated by sutures. Primary union resulted with a small discharging sinus at the base of the tongue which healed in two weeks. Under appropriate treatment, the red blood cells increased in a few months to 4,000,000. Then the patient discontinued his medication and his red cell count has fallen to 2,500,000.

In spite of a low blood count, cachexia, and advanced age, this patient not only stood the mild electrosurgical procedure very well but made a quick recovery. By the routine surgical procedure extirpation of the tongue might have been indicated. The electric procedure, at first glance, appeared to be too conservative. However, the involved area was destroyed by coagulation, followed by its removal with the electric loop with a wide margin of healthy tissue. The cancerous tissue following coagulation was so distinctive in appearance and consistency that one could readily recognize the limits of the tumor and differentiate it from the normal tongue, which permitted us to save the tip of the tongue with ultimate good functional palliative results (Fig. 2). Although 11 months have passed since operation, there has been neither local nor regional recurrence.

This case is an example of the removal of the coagulated tumor masses followed by primary union of the wound edges.

CASE II.—*Extensive recurrent carcinoma (Grade 2) of the right chest wall* (No. 34–1359. Path. No. L0703–19906). Seven years ago this patient, a white female, 53 years of age, had a radical operation for tumor of the left breast. Four years later several small lumps appeared in the right chest wall. These could not be controlled by roentgen ray treatment and the patient was rapidly failing.

When she came under our care the entire right chest wall was covered with raised, red, firm, easily bleeding, partly ulcerated nodules. There was confluent, foul ulceration at the right side, the nodules being attached to the skin and underlying tissues over an area 8 by 10 inches. There were also numerous pink firm nodules on the left breast and right scapula. In the supraclavicular regions, slightly movable tumor masses could be palpated. Many firm nodes could also be felt in the cervical region, up to the angle of the jaw (Fig. 3).

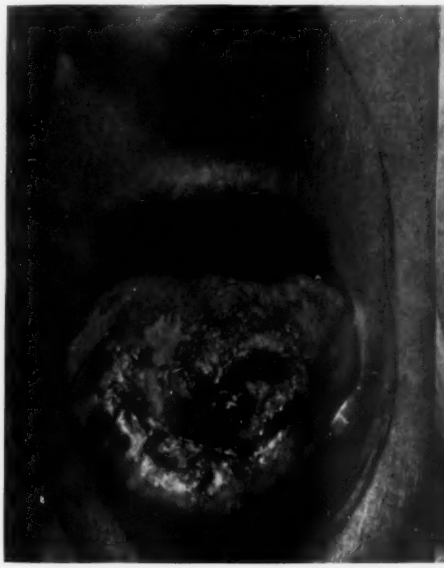

FIG. 1.—(Case 31–2247.) Squamous cell carcinoma of the tongue before electrosurgical treatment.

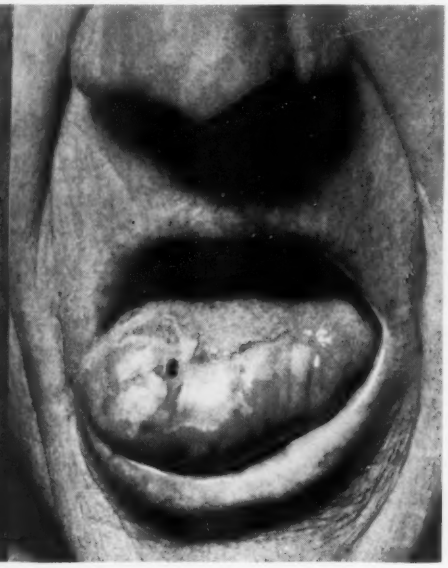

FIG. 2.—(Case 31–2247.) Squamous cell carcinoma of the tongue after electrosurgical treatment.

Under gas anesthesia, a partial cervical node dissection was done by electrosurgery. The lesions on the chest wall were removed by coagulation in four stages and the granulating wounds were Thiersch grafted. In spite of the large operative area, the patient stood the intervention very well.

This case illustrates the possibilities with electrosurgery, in cases where operative procedure would be difficult and radiation therapy a failure. It is 12 months since the original operation, but the patient has no pain or discomfort whatsoever and roentgenographic examination has not shown internal metastases. She is still under treatment and if new nodules appear in the skin they will be removed under local anesthesia (Fig. 4).

This patient represents a group of cases in whom electrocoagulation is followed by epidermis transplantation to the secondary granulating surface.

FIG. 3.—(Case 34-1359.) Extensive recurrent carcinoma of the right chest wall before electrosurgical treatment.

FIG. 4.—(Case 34-1359.) Extensive recurrent carcinoma of the chest wall after electrosurgical operations and Thiersch graft.

CASE III.—*Advanced carcinoma of the breast (adenocarcinoma Grade 2)* (No. 33–868. Path. No. 18718). A colored female, 75 years of age, first noticed a lump in her left breast four years ago. The lesion slowly increased in size and several months ago became ulcerated, discharging some blood.

When she came for treatment, her deformed left breast showed an oval, ulcerated, stony-hard mass, involving the entire outer quadrant. The tumor was attached to the surrounding tissues and underlying muscles. A few firm nodes were palpable in the axilla. Because of the advanced condition, operation was considered inadvisable. Palliative radium therapy, however, was administered for two months, totalling 2,912 mgh., with some improvement. Electrocoagulation was then advised for the persistent tumor.

The patient presented a foul, sloughing, secondarily infected mass (Fig. 5), involving the whole upper-outer quadrant of the breast and destroying the nipple. Scattered about the tumor were secondary areas of skin involvement. The primary tumor and the other involved areas were gradually eliminated by being first coagulated, then removed by the electric loop in order to sterilize the operative field. After another

FIG. 5.—(No. 33–868.) Advanced carcinoma of the left breast before electrosurgical operation and application of radium.

FIG. 6.—(No. 33–868.) Advanced carcinoma of the left breast after electrosurgical removal of growth and plastic operation.

preparation of the patient, the pectoralis muscles were partially resected and the axilla cleaned. The involved periosteum and intercostal muscle were superficially coagulated and carbonized. The area was covered with a skin flap measuring about five by nine inches, taken from the epigastric region, and after a drain was inserted, the wound was sutured (Fig. 6). The patient made an uneventful recovery except for a small area of necrosis of the skin flap which healed with secondary granulations.

This case illustrates the good result that may be achieved by electrosurgical methods in a case of far advanced, ulcerated, infected cancer of the breast when followed by an immediate plastic operation on the coagulated area, resulting in undisturbed healing. It demonstrates further the possibility of worthwhile palliation in a case ordinarily regarded as hopeless. In spite of the advanced lesion and the skin metastases, the patient is well, without evidence of local or regional recurrence 16 months after the plastic operation following sterilization of the infected area by electrosurgery.

CASE IV.—*Advanced squamous cell epithelioma (Grade 2) of the forehead* (No. 33–480. Path. No. 17751–17985–18033). A white female, 74 years of age, came to Stuy-

vesant Square Hospital with a history of "black spots" on her face for the last 18 to 20 years. One year ago a growth appeared on the right side of the forehead, rapidly increasing in size and gradually becoming painful.

Because of the patient's poor general condition—she had lost considerable weight—operation was not advised and a total of 160 mgh. of radium was administered by the insertion of platinum needles. Although there was slight improvement during the next two months, the frontal lesion persisted.

Physical examination revealed a poorly nourished, pale woman with chronic bronchitis. Red blood cell count, 3,460,000. The entire face was covered with hyperkeratotic areas. On the right side of the forehead there was an elevated, ulcerated, easily bleeding, necrotic mass two and one-half inches in diameter, surrounded by inflamed skin (Fig. 7). Roentgen examination showed that the lesion was attached to the underlying periosteum invading the frontal bone.

FIG. 7.—(Case 33–480). Squamous cell carcinoma of the forehead and hyperkeratosis of the face before electrosurgical treatment.

FIG. 8.—(Case 33–480.) Squamous cell carcinoma of the forehead and hyperkeratosis of the face after electrosurgical treatment.

The cancerous area was coagulated and removed (Fig. 8). The frontal sinuses were opened and cleaned out. The right frontal sinus showed cancerous involvement. This was coagulated and removed. The wound was packed with vaseline gauze. The patient's temperature was slightly elevated the first few days, but she remained in good condition and had no pain. The wound was clean after ten days, granulating except for the coagulated bone tissues. After three weeks there were healthy granulations with the exception of bone sequestration. There was no evidence of recurrence. Plastic operation* was performed after the necrotic bone was chiseled away. The wound edges were freshened and covered by two winged flaps, prepared from the front and united in the midline. A drain was inserted under the flap through the nose. The area

* The author wishes to thank Dr. Gustav Aufricht for his valuable advice concerning the plastic procedure.

from which the skin flaps were prepared was covered with Thiersch graft from the leg. Two days later the drain was removed and undisturbed healing took place.

Three months later we carried out electrosurgical treatment of the extensive hyperkeratotic areas. A new growth, also, was removed from the left eyebrow. Healing occurred with slight secondary infection and resulted in smooth, soft scars.

Judged by ordinary standards of radiology and surgery, this patient would have been considered a hopeless case, nevertheless, a worthwhile degree of palliation has been obtained in a relatively short time by electrosurgery. Twenty months have elapsed since operation and she is well, without evidence of either local or regional recurrence.

This patient represents a fourth group of cases in whom a secondary plastic operation is done following the original electrosurgery, at the indicated time (which is at least three to six months later), according to the type of tumor, unless there is indication for an earlier closure.

SUMMARY

Although a permanent cure may not be possible in many of the far advanced cases, patients may benefit by the amelioration of pain and by the elimination of a sloughing ulcerated carcinoma, which is very important not only from the individual but also from the social point of view.

Having had the opportunity to operate on a large number of advanced carcinomata in the New York Skin and Cancer Hospital, using the electrosurgical procedures described, we have been impressed by the relatively simple and mild character of the operation and its lower mortality. Patients are temporarily benefited even when distant metastases are present. Cachectic patients may be relieved of pains and the infected necrotic areas are eliminated. The four cases reported have demonstrated the palliative results following four different types of procedure.

The author wishes to express his appreciation to Dr. Henry H. M. Lyle and to Dr. Carl Eggers for their cooperation and the encouragement shown to him in this work.

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LEPTOTHRICOSIS

PULMONARY ABSCESS AND FATAL PYEMIA

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HUMAN infection from the filamentous forms of the high bacteria is rare and principally due to the actinomyces. The existing chaos regarding the whole subject of the higher bacteria is well shown by the many classifications which have been advanced.[1, 2, 3, 4] Mackenzie's classification is probably the most practical[5]:

CLASSIFICATION OF HIGHER BACTERIA (Mackenzie)

A. Typically filamentous forms ..... Actinomycetaceae
  B. Mycelium and conidia formed ..... Actinomyces
  BB. No true mycelium
    C. Cells show branching
      D. Gram-negative ..... Actinobacillus
      DD. Gram-positive ..... Erysipelothrix
    CC. Cells never branch: gram-positive: threads later fragmenting into rods ..... Leptothrix

A review of the literature on leptothrix infection shows it to be very rarely pathogenic for man; indeed, a surprising number of authorities[2, 3, 4, 13, 14] consider it non-pathogenic. Mackenzie[5] states that: "The great mass of bacteriologic literature is concerned with the species which inhabit the mouth and adjacent ducts and cavities: clinical and pathologic observations are related, as a rule, to diseases of the mouth and sporadic and unusual cases of meningitis, many or most of which are probably the result of infection by a variant of the bacillus of influenza." Harris[6] has recently reviewed the literature, collected five cases of severe human leptothrix infection, and emphasized its rarity. Two other reports[11, 12] are herein added to Harris' collection:

Author	Location
Harris[6]	Liver—multiple abscesses
Deussing[7]	Upper respiratory: kidney
Roedelius and Brack[8]	Upper respiratory: kidney
Von Meyenburg[9]	Liver
Lang[10]	Liver
Dick, Dick, and Rappaport[11]	Kidney
Thjotta[12]	Lung: bone

CASE REPORT

Hartford Hospital History No. 250004.—T. N., male, 23 years old, steelworker, was admitted June 20, 1931, complaining of cough and foul sputum of six months' duration. In October, 1930, he had been in an automobile accident suffering fractures

of both femurs and the right mandible. December 24, 1930, three weeks prior to the onset of cough, he had been a patient at the Norwegian Lutheran Deaconesses' Home and Hospital in Brooklyn, New York, where an abscess of the right jaw was incised and drained. Four days later he was discharged. There were no subsequent jaw symptoms. During the next three months he lost weight and strength, and the cough became productive of increasingly large amounts of sputum. Three months of hospitalization followed, during which time he received postural drainage and vaccine treatment for lung abscess. Two massive hemorrhages occurred during this period and at admission to the Hartford Hospital he was raising 500 cc. of thick, foul, greenish sputum daily and had lost 11 Kg. in weight.

FIG. 1.—Right lung, pleural thickening with drained abscess lower lobe.

FIG. 2.—Cut section of right lung: multiple abscess cavities.

Examination showed a moderately emaciated white male with a distressing foul productive cough, evidence of anemia, clubbing of the fingers, and signs of abscess of the right lung. Roentgenograms revealed multiple lung abscesses with associated pleural exudate on the right. Sputum examination showed mixed flora with no tubercle bacilli, no leptothrix reported. Blood studies substantiated the anemia; stool and urine examinations were not remarkable.

Two weeks after admission, under novocain anesthesia, rib resection was done and through an adherent pleura a large abscess cavity was deroofed and packed with gauze. Unfortunately, no culture was taken. Some improvement followed but he gradually failed despite blood transfusions and other supportive treatment. Six weeks after

thoracotomy signs of right psoas irritation were found. A large retroperitoneal abscess in the right perinephric region was drained, cultures showing a leptothrix in pure culture. Dextrose broth demonstrated long filamentous forms of bacteria with no branching. No growth was obtained on blood agar, aerobically or with reduced oxygen tension, or on anaerobic banana broth. The organism was recovered from an inoculated mouse but could not be subcultured. A few days later an abscess in the right forearm was evacuated, a similar leptothrix being obtained which, however, would not grow on blood agar, banana broth, or deep dextrose agar. Inoculation showed it non-pathogenic for mice. The subsequent course was one of gradual failure, with increasing emaciation, and death December 13, 1931, 11 months after onset.

Postmortem Examination by Dr. W. M. Allen.—*Gross Description.*—Adult white man of about 25 years showing marked emaciation. Small shotty nodes palpable in neck, axillae, and inguinal regions. Open surgical incisions in the lower midaxilla and in the posterior axillary line on the right, the first extending simply into the subcutaneous tissue to an irregular abscess cavity about 5 by 3 cm., the latter through partial resection of the ninth rib into the pleural cavity. A small sinus tract on the volar surface of the right forearm with an underlying cavity containing a small amount of exudate.

Thorax: Right pleura practically obliterated by rather dense fibrous adhesions. A small ragged abscess cavity in the region of the thoracotomy, with no demonstrable communication with a bronchus. Right lung: 720 Gm. (Fig. 1). On section (Fig. 2) the entire lung riddled with cavities 1 to 2 cm. in diameter, angulated and elongated and showing a fairly marked interstitial fibrosis of the cavity walls with surrounding grayish consolidation. Purulent material in the bronchi, the mucosa of which is hyperemic. Bronchi in many cases seen running into abscess cavities. Left lung: 620 Gm., with no adhesions or cavitation but showing a number of barely palpable dark red areas of apparent consolidation, more marked in the lower lobe, with the appearance of ordinary lobular pneumonia. Mediastinal and peribronchial nodes on the right, moderately enlarged, homogeneous, and without abscess formation.

Abdomen: Ascites slight. Liver: 3,400 Gm., enlarged downward to the umbilicus and with adherent omentum in a number of areas. Bulging abscesses under the surface (Fig. 3), many almost perforating. Liver on section (Fig. 4), riddled with large irregularly shaped abscesses up to 6 cm. in diameter, filled with thick grayish pus, and with only slight fibrosis of the walls. The surrounding liver substance hyperemic, but the remainder of the liver parenchyma essentially normal. Spleen: 380 Gm., slightly softened, and of ordinary appearance except for abscesses about 2 cm. in diameter in the lower pole, similar to those found in the liver, and some apparently having broken through to form an abscess surrounding the lower pole of the spleen and the splenic flexure of the colon. Kidney: 400 Gm., cortex not sharply limited. Adrenals: diffuse induration without abscess formation. Abdominal lymph nodes: slightly enlarged, of homogeneous pale-buff color and without abscesses or granulomata.

Microscopic Description.—Heart: moderate cloudy swelling without inflammatory reaction. Right lung: abscesses of every size in various stages of development, most of them fairly chronic with some fibrosis of the walls and showing very considerable tissue destruction. The cellular exudate, at least one half of the mononuclear type, *i.e.* lymphocytes, plasma cells, and macrophages. Small irregular particles of amorphous material in the center of the abscesses. Left lung: essentially a bronchopneumonic type of lesion, although here, too, exudate largely mononuclear with some early tissue necrosis, possibly representing an earlier stage of the process seen elsewhere. Lymph nodes: considerably hypertrophied, with loss of follicular arrangement but without abscess formation. Splenic abscess: entirely similar to those in the lungs. Liver: very little fibrosis in the walls of the abscesses, the uninvolved areas showing considerable cloudy swelling and fatty degeneration. Kidneys: essentially normal. Adrenals: congested and edematous but structurally intact.

Bacteriology and Animal Inoculation.—Direct smears from the liver abscesses

FIG. 3.—Superior aspect of liver: multiple bulging abscesses.

FIG. 4.—Cut section of liver: multiple abscess cavities.

showed masses of gram-negative bacillary forms, many small and slender; numerous fairly large fusiform shaped organisms; fairly numerous irregularly staining long slender filamentous forms. Blood agar plates remained sterile. Banana broth grew a gram-negative bacillus on the second day, on the fifth day some gram-positive bacilli occasionally in long forms were found, and on the eighth day some gram-positive bacillary forms and some long thin leptothrix-like organisms were isolated. Subcultures produced no growth. Prolonged microscopic study of numerous sections, including bacterial stains of the tissues, revealed no definite fungi.

Two cc. of the purulent material from a liver abscess were mixed with 10 cc. of infusion agar at body temperature and injected into a rabbit, 3 cc. into pleural and peritoneal cavities, and 1 cc. subcutaneously into the back and right side. One cc. of the unmixed pus was injected into the left side. The rabbit lived two months and autopsy showed a very extensive foul subcutaneous purulent infection burrowing into the inguinal regions. No abscesses or agar masses could be found in the peritoneal or pleural cavities, and no organism could be demonstrated or cultured from the foul abscess material. A guinea pig was injected with 0.5 cc. of the pus mixture into the pleural and peritoneal cavities. The guinea pig lived six months and at autopsy showed no sign of infection and no organisms.

Postmortem Diagnosis.[15]—Abscess of lung, right, due to actinomyces (leptotrichia) 360–202.2. Empyema, right 370–202. Generalized actinomycosis (leptothricosis) 012–202.

Discussion.—Bacteriologic studies are so varied that a complete review of the subject of the higher bacteria would serve to confuse the picture. The work of Mackenzie,[5] Wherry and Oliver,[16] Bulleid,[17] and Harris,[6] are worthy of note. The essential characteristics are that the leptothrix is an unbranched filamentous bacterium of the Actinomycetaceae group, varying in its staining qualities, difficult of culture, and rapidly losing its pathogenicity in successive animal inoculations.

Sufficient cases have not been reported to warrant any conclusions as to the value of forms of therapy other than that of any pyogenic infection. Dick, Dick, and Rappaport[11] report favorable results from the use of vaccine. Page,[18] in studying streptothrix infections, found vaccines of little value. Wangensteen[19] has recently cast considerable doubt on the value of the iodides and roentgen ray therapy in the treatment of infections due to the actinomyces. Graham,[20] in his recent text-book, does not include the treatment of leptothrix infections *per se* but summarizes the treatment of actinomycosis as follows: "Most of the successful treatments have included massive doses of iodides, roentgen ray therapy, surgical attack and autogenous vaccines. In several cases all three plans of treatment have been used." From available reports there is little uniform enthusiasm for any form of treatment except as an adjunct to surgery.

SUMMARY AND CONCLUSIONS

(1) Fatal leptothrix infection is exceedingly rare, as shown by a review of the literature.

(2) A case report with autopsy findings of lung abscess and metastatic abscesses of the spleen, liver, retroperitoneal tissues and forearm is presented.

(3) The finding of pure cultures of leptothrix in the drained retroperi-

toneal and forearm abscesses, and the obtaining of leptothrix from culture from the liver abscesses postmortem, warrants the diagnosis of leptothricosis.

(4) Treatment is that of any pyemic infection. Roentgen ray, vaccine, and iodide therapy have no proven value in leptothrix infection, but experiences in the treatment of infections with the other higher bacteria suggest further trial of these measures.

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FRACTURES OF THE JAW AND ALLIED TRAUMATIC LESIONS OF THE FACIAL STRUCTURES

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"Surgery to-day is being practiced by too many light-hearted and incompetent surgeons," says Sir Berkeley Moynihan.[8] Not infrequently jaw fractures and associated injuries of the bones of the face result in permanent displacement and deformity. The importance of reducing these fractures from a functional and from a cosmetic viewpoint is obvious. It is the duty of the surgeon with his knowledge of the mechanics and of the reactions of the tissues involved to prevent or, at least, to minimize these deformities. Extensive hospital experience with this type of work is unquestionably of the utmost value. While the specially trained oral and orthopedic surgeon is best fitted to handle these cases, accompanying crushing facial injuries involving vessels, nerves and muscles may present problems requiring the cooperation of the otolaryngologist, the plastic surgeon and the neurosurgeon. As a consequence, no doubt, of this mad machine age, the increasing number of these serious facial injuries demands a careful consideration of the methods which constitute the most satisfactory forms of treatment.

Location of Fracture.—When considering the location of fractures, we find that the rami, condyles and coronoid processes, protected somewhat by their anatomic placement, are less frequently broken, while the vulnerable position of the mandible causes its involvement in a very large percentage of facial injuries. The point of fracture is determined not only by the strength and thickness of bone but by the direction from which the traumatizing blow is received. In the inferior maxilla we find the symphysis to be the strongest portion, the mental foramen, the weakest, and the angle, though heavy and apparently resistant to trauma, to be one of the most frequent points of fracture.

Reduction Method.—The voluminous literature on the subject of jaw fractures covering, as it does, many and varied types of technic and treatment, shows plainly the lack of uniformity of opinion. Effective measures and devices have been originated or modified by G. V. I. Brown, G. M. Dorrance, H. S. Dunning, T. L. Gilmer, V. H. Kazanjian, Chalmers Lyons, Sterling Mead, F. B. Moorehead, R. T. Oliver, Kurt Thoma, and Leo Winter. It was Doctor Gilmer[3] who pointed out the value of wiring the mandibular teeth to those in the superior maxilla for fixation in fractures of the mandible. Ivy and Curtis[4] describe a modification which they consider superior since it permits a greater degree of accessibility to the mouth for replacement of a broken wire, *etc.*, without disturbing the main attachments on the teeth. From per-

sonal experience, we consider that our results from the use of the Ivy-Curtis technic of eyelet wire fixation have been sufficiently favorable to warrant its uniform adoption.

Synopsis of Case Series

Table I

General features—sixty cases

Fracture Cases	No.	Per Cent
Sex		
Male	43	71.7
Female	17	28.3
Type		
Single (simple)	7	11.7
Single (compound)	35	58
Double (compound)	15	25
Triple (compound)	3	5
Site		
Through maxillary incisors	2	3.3
Depressed malar bone	6	10
Depressed zygomatic arch	3	5
Condyloid process	4	6.7
Coronoid process	1	1.7
Ramus	2	3.3
Angle of mandible	15	25
Region of mandibular molars	20	33.3
Region of mandibular premolars	4	6.7
Median line of mandible	3	5

Table II

Age distribution—sixty cases

Fracture Cases	No.	Per Cent
10–19	10	16.7
20–29	22	36.7
30–39	12	20
40–49	9	15
50–59	4	6.7
60–69	2	3.3
70–79	1	1.7

Table III

Causes—sixty cases

Fracture Cases	No.	Per Cent
Automobile accidents	36	60
Fights	21	35
Falls	2	3.3
Gunshot wound	1	1.7

Cause of Fractures.—In respect to the principal causes of mandibular fractures, we find that Dunning[2] quotes 55 per cent of 1,065 cases as having been due to assault and battery; in a series of 100 cases, Ivy and Curtis[5]

list 49 per cent under assault and battery; Winter,[9] in a series of 200 cases, assigns 75 per cent to the same cause. In connection with our own series, the listing of automobile accidents as the most frequent causative factor seems somewhat at variance with the findings in eastern localities. We venture to suggest that the reason is to be found in the extensive automobile highways throughout the west and their too widespread use at dangerous rates of speed. On the other hand, the huge metropolis with its polyglot throngs would almost certainly have a preponderance of injuries actuated by the more usual forms of physical violence.

DIAGNOSIS.—A careful history as to the nature and time of the accident should be obtained. The diagnosis in jaw fractures presents no difficulty in the presence of abnormal and painful movement, displacement, loss of function, malocclusion of the teeth, and, most important of all, roentgenographic examination, at least two views. These are particularly valuable for showing the direction of fracture lines and the relationship of fragments and tooth roots. Further, the roentgenogram affords an indisputable record in medicolegal complications. According to Ivy and Curtis,[4] "in every case of fracture in the region of the mental foramen, we should always look for a second fracture at the angle on the opposite side."

In a fracture through the neck of the condyle, or ramus, there is shortening of the distance from the angle to the glenoid fossa on the affected side, so that the mandible drifts to the opposite side causing the molars on the injured side to meet before those on the opposite arch. In fractures through the mental foramen, the anterior fragment is displaced or drawn downward by the action of the depressor muscles, while the posterior fragment is pulled upward by the masseter, temporal and internal pterygoid muscles. In a fracture through the angle, the lower end of the short fragment is generally displaced upward and forward by the contraction of the elevator muscles. The larger fragment frequently is drawn slightly toward the fractured side. In mandibular fractures, the displacement is contingent upon, and governed by, the action or interaction of the muscles to which the jaw gives attachment. We may add, too, that a comprehensive understanding of muscle function and control is of inestimable value.

TREATMENT.—While the slogan in every fracture case may be said to be "early fixation," mandibular fractures must sometimes prove an exception. Immobilization of the jaws should not be attempted until the general condition of the face and mouth is such that the fixation may have a reasonable expectancy of remaining in place. The factors to be considered at this time are: (1) edema of the soft parts; (2) mouth sepsis; (3) removal of carious roots and loosened teeth, especially along the line of fracture, with the attendant possibility of hemorrhage; and, (4) necessary attention to the remaining teeth from the standpoint of an additional source of infection.

In the presence of edema of the soft tissues, the application of hot and cold compresses is valuable. Preparatory to coaptation of the bony parts, the eradication of mouth sepsis cannot be stressed too strongly, since the end-

result is almost entirely dependent upon the degree of cleanliness that it has been possible to maintain in the mouth. The presence of carious teeth and roots, of course, constitute a bacterial menace which should be eliminated. However, the retention temporarily of a loose or decayed tooth may be justifiable in cases where a fragment would become edentulous by its removal.

Temporary Support.—Where immobilization had to be delayed, we found that a temporary head support or bandage not only gave the patient a sense of comfort and security but increased his willingness to cooperate. In using this support, the teeth are fixed in fairly good occlusion, a small abdominal

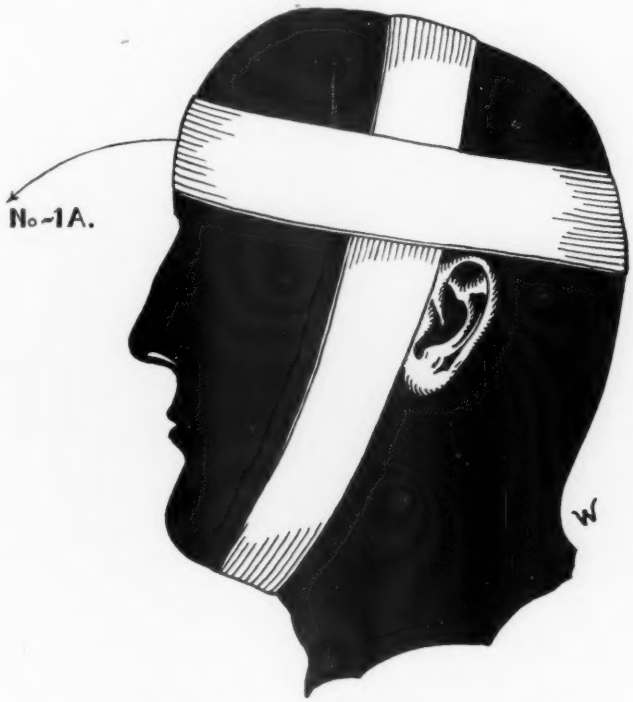

FIG. 1.—Modified Gibson bandage for support of fractured jaw.
1A.—Reversal of roller at left temple.

pad is placed under the chin, and, using two inch muslin, a modification of the Gibson bandage is applied. From the vertex of the skull the roller passes downward in front of the right ear, under the chin and up in front of the left ear to the starting point. This is repeated three times, then, at the left temple, the bandage is reversed and carried horizontally around the head three times. Adhesive strips, half an inch narrower than the bandage, are then placed over it (Fig. 1).

Immobilization Method.—Regardless of the situation in the jaw, about 90 per cent of mandibular fractures are reduced efficiently by the eyelet wire technic of fixation. We have already mentioned our preference for the Ivy-Curtis[4] method which is, briefly: the teeth selected for wiring must be located opposite each other on the jaws; when available the premolars and

incisors are used. Six eyelets in pairs are used; these are made by taking six inch strands of No. 24 gauge soft brass wire, forming a small eyelet in the center of the strand and retaining it by a double twist. With the eyelet held on the buccal surface, the ends of the strands are passed between the teeth selected, bent in opposite directions, and fitted firmly and snugly around the teeth in order to hold the eyelet in a fixed position. All excess wire is cut off and the ends tipped with modeling compound to protect the mucosa. Through each of the three pairs of eyelets is passed a twelve inch strand of the same wire. The patient is asked to cooperate with the surgeon in bringing the jaws together as closely and naturally as possible. With the twisting and tightening of the three strands, complete immobilization is obtained (Fig. 2).

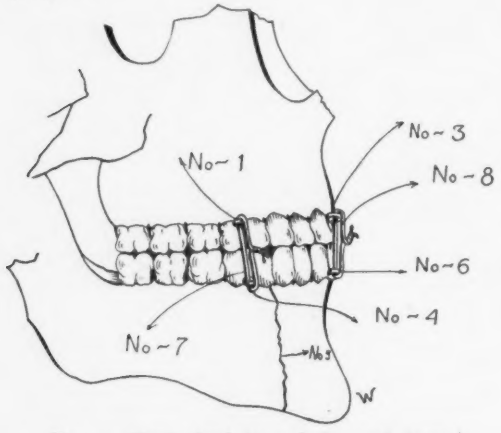

FIG. 2.—Teeth fixed in occlusion and jaws immobilized. Nos. 1, 3, 4, 6.—Eyelets. Nos. 7, 8.—Single tie wires through maxillary and mandibular eyelets. No. 5.—Line of fracture.

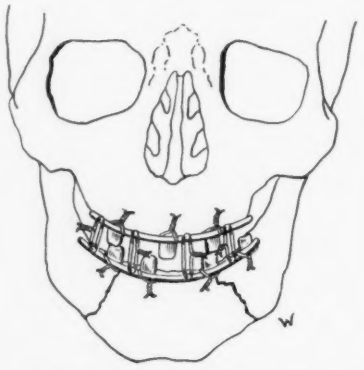

FIG. 3.—Fixation using modified half round arch bars (after Ivy-Curtis) where insufficient teeth prevent the use of eyelet technic.

General Considerations.—In the absence of opposing teeth suitable for eyelet fixation, we used half round arch bars (modified) as recommended by Ivy and Curtis[4] (Fig. 3). We found it expedient to maintain the temporary support for approximately one week after fixation, removing it periodically for necessary facial hygiene. For irrigations of the oral cavity, we used Carrel-Dakin therapy and a saturated solution of sodium perborate alternately.

Duration of Fixation.—Definite periods for fixation cannot be established since accompanying factors are rarely if ever identical. However, in our uncomplicated single fractures, the average time for immobilization was three weeks; in double compound, uncomplicated fractures, thirty-two days. Fixation periods designated by other surgeons are: Dean,[1] 43 days; Ivy and Curtis,[5] 31 days; Rogers,[7] 28 days; Winter,[9] 36 days. The same authors point out, though, that with complications the period may extend from nine to 18 weeks.

COMPLICATIONS.—The complications of jaw fractures are chiefly those of infection. Where a root or spicule of bone is present in the line of frac-

ture, it is advisable to provide external drainage at the time of immobilization (Fig. 4). Abscess formation is not unusual, and the pus often gravitates into the submaxillary, cervical or sublingual spaces. This is best drained by making a small incision through the most pendulous part of the mass, then, with a hemostat, doing blunt tunneling after the Hilton method. Consideration should be given to the cosmetic result, to the anatomic structure, and to the avoidance of traumatizing the bone. Very often this suppuration affects

FIG. 4.—Example of detached spicule of bone in line of fracture. Failure to establish drainage resulted in bone destruction and delayed union.

only the soft tissues and will clear up promptly, but, should it persist unduly, osteomyelitis is indicated.

Osteomyelitis.—According to the literature, osteomyelitis is not an unusual complication in jaw fractures. This condition is suspected by constant and excruciating pain and is located definitely by gently tapping the affected bone. In the early stages, the roentgenogram is of little value since bone destruction does not occur for at least three weeks after infection. The

alveolar processes in an infected area usually undergo a degree of degeneration that affects the teeth. During the acute stage of the disease, many surgeons are emphatic in their protests against any form of operative procedure. Chiseling or curettement of the diseased parts not only destroys the growing bone tissue but endangers the nutrient artery. Exfoliation of sequestrum by natural processes, and its removal through the mouth when possible, will result most favorably. Hot wet local dressings should be used freely, as well as mouth irrigations of potassium permanganate (1:4,000), or Dakin's sodium hypochlorite solution. Maintenance of the patient's general condition should not be overlooked.

In our series of mandibular fractures, ten (all males) were complicated by infection, and six of these developed an osteomyelitis. In the subperiosteal abscess cases, healing and union required from 40 to 50 days, while those complicated by osteomyelitis needed from 65 to 90 days.

Malunion.—Late and improper treatment is nearly always responsible for this condition. Its most serious complications are malocclusion and dysfunction. Recourse to osteotomy is usually necessary for repair.

Delayed Union.—In delayed union we are confronted with an abnormal time element although union is constantly progressing. A factor may be a systemic calcium-phosphorous deficiency. Local treatment, especially in regard to the reposition of the fragments, seems the best stimulant for satisfactory union.

Non-union.—When, for any reason, normal osteogenesis is prevented, reparative processes cease. As far as we know, local causes of non-union are: suppurative processes, osteomyelitis and necrosis, traumatized blood supply, and too frequent manipulation of bony fragments; while general causes are: syphilis and tuberculosis, calcium-phosphorous deficiency, and age and general debility.

Fractures of Edentulous Mandible.—In our own limited experience with this condition, we were able to utilize the artificial dentures of the patient, first reinforcing the alveolar ridges with impression compound. When the displacement of fragments is marked, Ivy and Curtis[4] quote Black's suggestion of circumferential wiring of the bone.

Fractures of the Neck of the Condyle and the Coronoid Process.—In five cases, we successfully employed the Ivy-Curtis eyelet wire fixation technic. Immobilization averaged three to four weeks. Our cases did not result in ankylosis to a point of fixation of the joint, and temporary stiffness was treated with massage and physiotherapy.

Fractures of the Maxilla.—These fractures are usually the result of crushing facial blows that have injured adjacent structures at the same time. These complications, together with the delayed reduction of the fracture necessitated by them, may cause permanent facial deformities. The superior maxilla is not influenced by powerful muscle attachments that have a tendency to distort a fractured area, but lacerations and extensive edema may be considered a certainty, therefore roentgenologic study is invaluable.

Infection is not uncommon, but the rich blood supply to this region makes necrosis relatively rare. The fractured maxilla, being almost without exception merely one phase of the injury, must be considered from the viewpoint of the entire involvement in the matter of treatment.

FRACTURES OF THE MALAR BONE AND ZYGOMATIC ARCH.—Located so prominently, the malar bone and zygomatic arch assume a significant position among the bones of the face, particularly in regard to cosmesis. All of the facial structures are important, not alone through their external distinctness but because both they and their foramina, attachments, and borders involve an area of very complicated and vital structures. Fractures in this region, usually the result of extreme direct force, should be reduced as soon as possible, since delay increases the difficulty in approximating the bony parts.

In the determination of these fractures, the surgeon will obtain valuable aid from direct palpation both intra- and extrabuccal. The latter, in particular, is both simple and efficient for the zygomatic arch is readily palpated in the average person and even more clearly defined in those afflicted with any wasting disease.

Reduction Method.—Many and ingenius methods have appeared in the literature from time to time for the elevation of these fractures, one being a method originated by Matas.[6] In several of our own cases, which included fractures in the malar bone as well as those in the zygoma, we applied a modification of the Matas technic (Fig. 5), with such completely satisfactory results that we outline it briefly: the skin and adjacent soft parts to the fracture in the bone are infiltrated with a 1 per cent solution of novocain. An absence of muscle tension is desirable, and this can be obtained best by asking the patient to keep his mouth open about one-half an inch. The area immediately in front of the coronoid process (the continuation of the anterior border of the ramus) is palpated against the infratemporal surface of the maxilla until the masseteric border of the malar bone is engaged. At this point, which is directly in front of the coronoid process, a tiny superficial incision is made to accommodate the needle, or, if the area be outlined very clearly, an incision is unnecessary. A large sized Hagedorn needle, or one of similar design and function, is introduced at the site of incision, passed carefully beneath the depressed

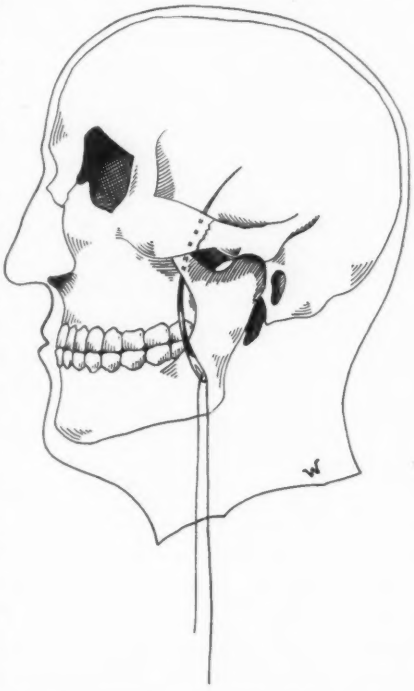

FIG. 5.—Authors' modification of the Matas method applied to depressed malar fracture.

fragment, and directed to emerge at a point just beyond the lateral portion of the temporal border of the malar bone. The needle is threaded with No. 22 gauge copper wire, and the latter passed through sterile petrolatum preparatory to entering the skin. The two ends of the wire are twisted together in one direction and traction made upward and slightly medially on the depressed fragment in order to bring it into normal position. At times the bone assumes its correct position with a sucking noise (like that of a boot withdrawn from mud), and a final click. More often, however, no sound is heard and considerable traction must be exerted before replacement is accomplished. After the reduction has been achieved, the incision is closed with a single fine suture. In these cases we have been able to dismiss the patient within 48 hours.

Recurrence of the deformity is rare and trismus, unusual. In simple fractures involving both malar bone and zygomatic arch, where the zygomatic-temporalis suture is depressed but not demarcated, it is possible to elevate both bones at one operation. Where circumstances permit, this mode of treatment will, we believe, prove simple and satisfactory, and will obviate the difficulties and objectionable features of more radical surgery.

RÉSUMÉ

In cases of this type, due to trauma, it may be assumed that the individual had been enjoying a fair degree of health. Therefore, in addition to the proper handling of the traumatic involvement, there is the twofold objective of restoring the patient to his usual vocation with all consistent speed and with the least possible impairment to his previous state of health.

Necessary considerations regarding the fracture are: (1) Regional involvement that will affect directly the reduction of the fracture. (2) Extreme precaution in preventing or minimizing infection. (3) Early reduction of the fracture. (4) Postoperative examination, at least periodically, until final discharge.

In the general handling of the patient: (1) We found local anesthesia to be especially valuable in fracture reduction, for it permitted cooperation with the surgeon during operative procedure, and eliminated the nervous dread of taking a general anesthetic as well as the systemic after effects which would tend to prolong hospitalization. (2) Attention was given to the need of an adequate fluid intake, and special diets were arranged. (3) We found physiotherapy invaluable as an aid both in the traumatized area and in connection with the general physical tone. (4) In mandibular fractures, good results were obtained by allowing the patient a temporary discharge from the hospital in order to pursue some form of light employment.

We wish to acknowledge our indebtedness and to express our appreciation to Dr. H. M. Ginsburg, Director of the General Hospital of Fresno County, Fresno, California, and to Dr. D. I. Aller, Chief of Orthopedic Surgery.

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THE NICOLA OPERATION FOR RECURRENT DISLOCATION OF THE SHOULDER *

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THE purpose of this brief paper is to describe the Nicola operation for recurrent dislocation of the shoulder joint, and to report the results of a short series of cases, which have been followed postoperatively over an average period of two and one-half years.

The number of methods which have been devised for the cure of recurrent dislocation of the shoulder joint is the best index of the difficulties of the problem that this condition has presented. The most outstanding of these may be quickly reviewed according to the structure utilized to correct the fault.

Capsule.—Riccard, Thomas, Bardenheur and others have reinforced the capsule in various ways, usually by plication of the anterior and inferior surfaces.

Muscle and Tendon.—Fensterer and Heyman transplanted the short head of the biceps to the upper rim of the glenoid fossa suturing the tendon along the anterior capsule.

Young lengthened the pectoralis major and latissimus dorsi.

Perthes reinserted the supraspinatous.

Clairmont dissected the posterior fifth of the deltoid from its origin and carried it through the quadralateral space under the joint, fastening it to the coracoid process anteriorly.

Schosserer and Cotton shortened the subcapularis.

Bone.—Chaput and Alibut resected the humeral head.

Hildebrand deepened the glenoid fossa from behind, making the anterior lip more prominent.

Eden, Mauclaire, Speed and Hybbinette placed a bone graft along the anterior margin of the glenoid fossa.

Oudard, Lenormant and Muard elongated the coracoid process to form a block against the anterior displacement of the head.

Fascia and Tendon.—Payr and Kirschner, in 1913, were the first to employ fascial transplants to reinforce the shoulder joint.

Carrell divided the long head of the biceps. The distal end was sewed to the short head. The proximal end was elongated with fascia lata and was carried under the neck of the humerus and fastened behind through a drill hole, into the acromion.

Spitzy wove strands of silk around and through the capsule attaching them to the coracoid process.

* Read before the Joint Meeting with the Boston Surgical Society and the Philadelphia Academy of Surgery, on February 4, 1935, at Philadelphia, Pa.

Joseph and Gratz used fascia lata to make a ligament from the humeral head to the acromion.

Cattaneo used a sling of fascia lata around the capsule fastening the ends over the acromion.

Henderson transplanted the peroneus longus, threading it through the acromion and the greater trochanter of the humerus.

Wahl divided the long head of the biceps anchoring the proximal end into the greater trochanter and sewing the distal to the periosteum of the bicipital groove.

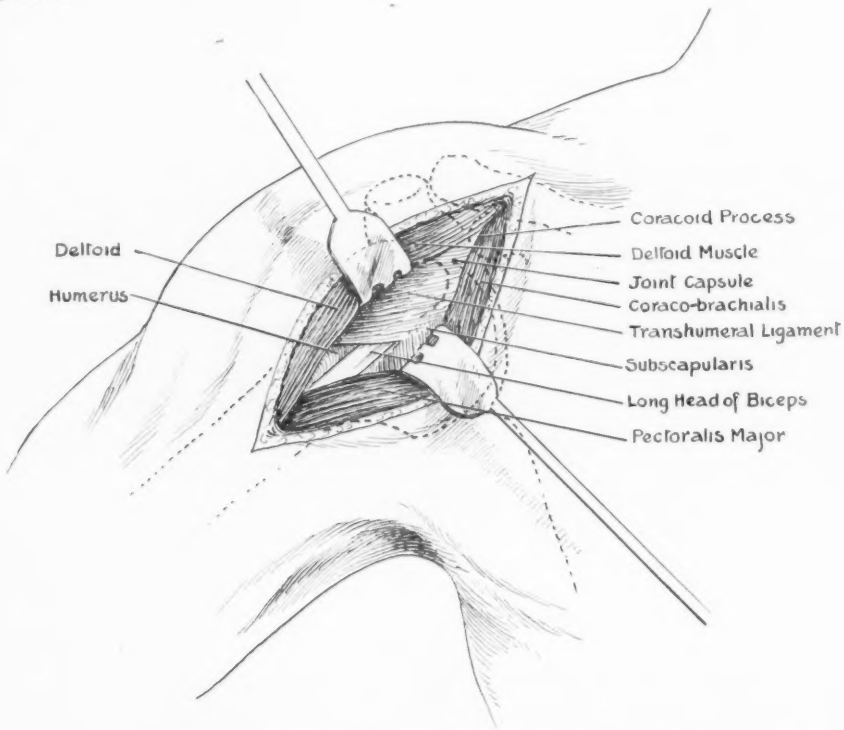

FIG. 1.—Showing approach between the deltoid and pectoral muscles.

Fowler advocated a fascial sling carried beneath the neck of the humerus and fastened anteriorly to the coracoid process posteriorly to the acromion.

Tuffic Nicola observed that the shoulder joint of most anatomic skeletons was fixed by a pin through the center of the humeral head to the glenoid fossa of the scapula. This method of fixation allowed practically normal range of motion. Recalling the similarity of the ligamentum teres of the hip joint, he conceived the ingenious idea of utilizing the long head of the biceps tendon as a check ligament to stop abnormal forward and downward motion of the humeral head on the glenoid fossa.

In 1929, he reported one case in which this method of stabilization was used. The technic of the operation consisted of the following steps:

(1) Incision from clavicle just above coracoid process four inches down along anterior margin of deltoid (Fig. 1).

(2) Division of long head of biceps one-half inch above the point where it disappears under the pectoralis major.

(3) Drilling of a hole in the bicipital groove, at level of lower edge of the transverse humeral ligament upward to emerge at the center of the head of humerus.

(4) Threading of the proximal end of the tendon through the drilled hole using a flexible probe.

(5) Resuture of the divided tendon under tension.

(6) Fixation of the arm by Velpeau bandage for three weeks.

In 1931, Nicola reported a series of 20 cases operated upon by various surgeons. The end-results were uniformly satisfactory.

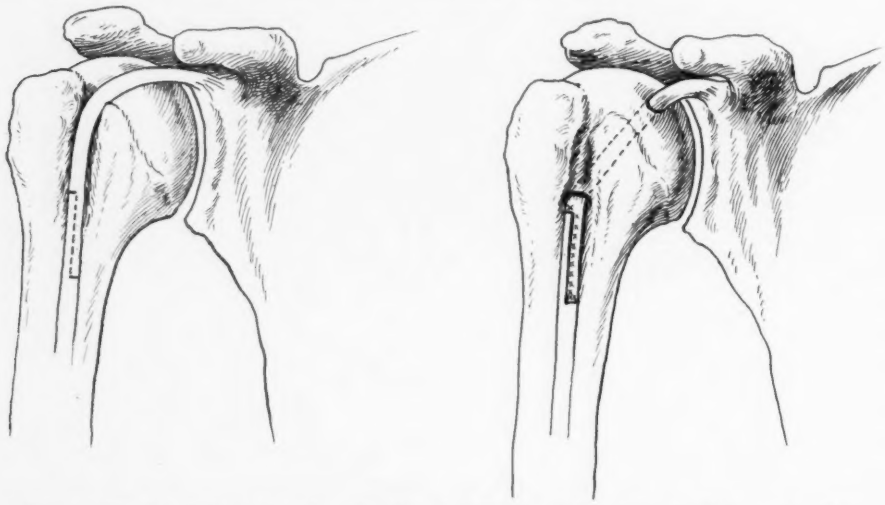

FIG. 2.—Showing division of tendon, direction of drill hole, and tendon sutured in new position.

The author had the opportunity of seeing Doctor Nicola's first case, and was impressed both by the simplicity of the technic and the functional efficiency of the result. Since then he has used this method with the following slight changes in procedure and after care.

(1) Z instead of transverse cut in tendon to give plenty of overlap for suture (Fig. 2).

(2) Placement of drill hole well anterior and above center of the articular surface of the humeral head to avoid anterior luxation and limitation of abduction.

(3) Placement of the arm in abduction with elbow flexed to right angle, while suturing the tendon to take up the slack of biceps tendon.

(4) Anchorage of the distal end of the proximal portion of the tendon to the periosteum at the point of emergence from the drill hole.

(5) Velpeau bandage for five days postoperatively and then a sling for remainder of three weeks.

(6) Passive extension and flexion of shoulder and elbow to be started at end of second week.

(7) At the end of third week, active circumduction exercises of the arm with the trunk bent forward, and with gradual increase in the arc of motion.

(8) Avoidance of elevation beyond the shoulder line for eight weeks.

In the ten cases operated upon, we have a follow up period of from one and one-quarter years to five years. The age of the cases at operation ranged from 16 to 45 years. Occupation was usually one of the more strenuous types, and, in each case, was resumed postoperatively. In the student group, there were three college men who played on varsity teams, football, basketball or crew. The one girl in the series resumed her tournament tennis. Of the laboring group there were two miners, a truckdriver, and two unemployed, who claimed they do strenuous work when the opportunity offers.

The end-results are graded according to the code accepted by the Fracture Committee. They are according to anatomic, functional, and economic results. The grade of 1 equals 25 per cent; 2 equals 50 per cent; 3 equals 75 per cent, and 4 equals 100 per cent.

There were two complications at the time of operation which bear mentioning. The first, in a case operated two days after last recurrence—a large vein presented itself in the bicipital groove. Exploration beneath failed to reveal the biceps tendon. Not without temerity this was opened to verify an hematoma of the tendon sheath. The second was a tiny thread of a tendon running from the muscle mass to the upper anterior aspect of the greater trochanter. Deeper exploration in the bicipital groove, however, disclosed the long tendon and proved this first to be a third bicipital tendon.

In the early cases slight postoperative subluxation of the shoulder joint was present, as the importance of abducting the arm and flexing the elbow was not observed. In each case, however, this slackness had been taken up by the end of three months, probably by contracture of the proximal portion of the tendon.

Case 4 had an old elbow fracture of the same arm with considerable limitation of motion. He had had the greatest number of recurrences. The dislocation could be produced by rolling over in bed. His joint showed hypertrophic bony changes. He has resumed his work in the mines, but has some aching in his shoulder after a day's work. He has been graded 3 for function.

Case 5 had a recurrence. This was a very muscular individual, a miner by occupation. Seventeen months after operation he was in a swimming race and struck a channel stake on the point of his shoulder joint at the top of his stroke. The dislocation was reduced. He has had no recurrence in the 16 months following, and has continued his work as a miner. He has, however, observed certain precautions. He was graded 1 for function.

Case 7, whose etiologic factor was epileptic convulsions, was held in a plaster Velpeau for six weeks. He has some anterior luxation of the joint when arm is abducted. He continues to have about 20 degrees limitation of abduction and external rotation. He was graded 3 for functional and 3 for

anatomic result. He cannot get a job, but the convulsions are probably a greater handicap than the shoulder (Table I).

TABLE I

Case	Age	Occupation	Side	1st Disloc.	Recurrences	Force	Operation	Follow Up	Result A.	F.	E.
1. T. W..	19	Student	L	1 yr.	4	Ab.-Ext.	1/31/30	5 yrs.	4	4	4
2. F. L...	16	Student	R	1 yr. 8 mos.	5	Ab.-Ext.	6/ 4/30	4 yrs. 6 mos.	4	4	4
3. M. S...	18	Student	L	4 yrs.	4	Ab.	12/19/31	3 yrs.	4	4	4
4. S. C...	24	Miner	R	1 yr. 6 mos.	17+	Ab.-Ext.	3/ 7/32	2 yrs. 9 mos.	4	3	4
5. W. G..	38	Miner	L	1 yr. 6 mos.	5	Ab.	3/24/32	2 yrs. 9 mos.	4	1	4
6. L. L...	28	Salesman	R	4 yrs. 6 mos.	12	Ab.	10/28/32	2 yrs. 3 mos.	4	4	4
7. A. B...	31	Laborer	L	1 yr. 20 mos.	7	Convuls.	9/ 5/32	2 yrs. 3 mos.	3	3	3
8. A. C...	45	Laborer	R	12 yrs.	12+	Ab.	6/ 6/33	1 yr. 6 mos.	4	4	4
9. G. S...	27	Truckdriver	L	4 yrs.	6	Ab.-Ext.	6/15/33	1 yr. 6 mos.	4	4	4
10. J. D...	23	Typesetter	R	2 yrs.	4	Ext.	8/24/33	1 yr. 3 mos.	4	4	4
Summary	27		5R-5L	3 yrs. 4 mos.	7+			2 yrs. 8 mos.			

SUMMARY

The operation advocated by Nicola for recurrent dislocation of the shoulder has been used by us in ten cases, with an average follow up of two years and eight months. The operative technic was the easiest of those devised for this condition. The postoperative convalescence and period of hospitalization were short. The results graded according to anatomic, functional and economic values were high. One case only had a recurrence due to an accident more than a year after operation. All cases returned to their former occupations subsequent to operation.

DISCUSSION.—DR. T. TURNER THOMAS (Philadelphia).—Few surgeons discuss operations for recurrent dislocations of the shoulder because few do them. I have done about 70 cases by the axillary route but none by these new suspension methods. In my last six cases I have tried something new which I think reduces the risk of recurrence.

I have a special interest in one of Doctor Willard's cases because I was present when he presented it before the Academy of Surgery a few years ago. It was the most detailed presentation of a case so operated on that I have heard, a good history, description of operation and the results afterward. This patient swung his affected arm around vigorously in all directions without difficulty and played football afterwards.

But Doctor Willard told us something that interested me more. He said that when the shoulder was in a severely strained position as in football, "it slid over but without a dislocation." That interested me very much because with the pathology not exposed or corrected I have been expecting to hear of some disturbance in the joint after this operation and suspected that it must occur, but Doctor Willard is the first to refer to it so far as I know.

My most serious objection to this and other operations like it is that it does not expose and consequently does not try to correct the pathology. The obvious reason is that the pathology is in the axillary portion of the joint and difficult to expose. Before these new methods came into vogue all operations aimed at restoring the joint to the normal and had a remarkable degree of success. If there were any deaths or bad results they were not reported. I cannot conceive of any of these suspension methods restoring the joint to the normal without removing the pathology although I can see how they may prevent the dislocations.

According to my conception of the mechanism and pathology of anterior dislocations of the shoulder there develops around the protruding head an axillary hernial pouch of capsule and synovial membrane which in recurrent dislocations is ever ready to receive the humeral head whenever the hyper-abducting force is strong enough to pry it out of the socket. The long head of the biceps in its transplanted position cannot hold it in the socket in this position of abduction which relaxes it. But after the head has "slid over" the glenoid margin into the dislocated position, the arm in dropping to the side of the body from gravity can tighten the biceps tendon, which can then pull the head back into the socket. It could provide a spontaneous reduction as probably often happens in unrecognized dislocations of the shoulder.

The success of this operation depends entirely upon the integrity of the transplanted tendon. How long it can withstand the wearing friction of its artificial bony canal has not been determined. The overwhelming majority of my cases have had for varying periods, some for 25 years, down to the last reports more or less recently, complete absence of the recurrent dislocations and apparently normal joints. There is nothing in the literature, evidently, to indicate that other surgeons by the older methods, except excisions, have not obtained as good results. More detailed reports will be necessary to establish as good results for the newer methods.

DR. DEFOREST WILLARD.—Doctor Thomas in his discussion brings out the fact that the Nicola operation does not correct the relaxation of the shoulder capsule. It does, however, prevent the humeral head from exerting force against the weakened capsule, providing that the insertion of the biceps tendon in the humeral head is correctly placed. I believe that prolonged fixation of the head by this new tendon allows the weakened capsule to thicken and strengthen. Although we had a little sliding, in our earlier cases, this has now ceased, and our one recurrence was due to a major accident, which would probably have dislocated any normal shoulder.

VOLKMANN'S ISCHEMIC CONTRACTURE IN HEMOPHILIA

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IN 1822, Volkmann[1] described a contracture of the muscles of the leg in a 16 year old boy which followed the application of a splint and a compression bandage in the course of treatment of hydrops of the knee joint. This description is the first record of the recognition of a comparatively clear cut clinical picture now generally known as Volkmann's ischemic contracture. A particular interest in the etiology of this condition was stimulated by the studies of Bardenheuer,[2] in 1911, and J. B. Murphy,[3] in 1914. Whereas Volkmann[1] and Leser[4] had emphasized anemia from arterial obstruction as the principal causative factor, Bardenheuer and Murphy believed that obstruction of the veins was the important etiologic agent. From experiments on animals, Brooks,[5] in 1922, and Middleton,[6] in 1930, showed that an acute myositis and subsequent fibrosis and contracture of a single muscle could be produced by venous obstruction. These experiments would seem to substantiate beyond reasonable doubt that the characteristic clinical picture and pathologic changes of Volkmann's contracture could be reproduced in animals by venous obstruction. The fact remains, however, that numerous clinical observations, for example, those of Tinel,[7] and experimental evidence from the studies on arterial obstruction, for example, Brooks and Johnson,[8] make it impossible to avoid the conclusion that fibrosis and contracture of muscles may also be produced by arterial obstruction. A clear cut differentiation of the contractures following arterial obstruction from those following venous obstruction has not as yet been made.

The relationship between the application of a constricting dressing and the subsequent development of Volkmann's contracture has frequently been of more than academic interest to surgeons. Murphy[3] states: "The lesion in Volkmann's contracture is a surgeon's lesion and never a lesion due to the trauma. It is a lesion due to a defective dressing." It is interesting to note, however, that two years after the publication of this statement, Murphy[3] reported an instance in which Volkmann's contracture had occurred without a previous constricting bandage. He states: "Certain it is that a severe contusion of the muscles of an extremity without fracture or external wound or evidence of injury may be followed by this contracture." Other instances of Volkmann's contracture without the previous application of a constricting bandage or of a fracture have been recorded. The purpose of this paper is to

report an instance of the development of Volkmann's ischemic contracture in a hemophiliac in whom no bandage or splint had been used and in whom there was no preceding trauma. Five such cases have been previously reported.

Case Report

A 22 year old white male was admitted to the Vanderbilt University Hospital February 19, 1927, complaining of pain and swelling of the knee and elbow. The patient's past history was characteristic of hemophilia. On numerous occasions he had bled profusely from a small wound. There had been frequent attacks of spontaneous ecchymoses in the skin, sudden development of pain and swelling in joints and bleeding from the gums. The patient had two living brothers who were similarly affected. A third brother died at the age of eight from a profuse hemorrhage from a slight blow on the nose. The patient refused to remain in the hospital for a sufficient length of time for complete examination. Blood examination, however, showed normal platelet count and approximately normal clotting time.

The patient was readmitted to the Vanderbilt University Hospital July 8, 1927, because of persistent bleeding from the gums. He again refused to remain for a complete examination. The previous pain and swelling of the joints had for the most part disappeared.

November 15, 1932, he was admitted to the Vanderbilt University Hospital for the third time because of contracture of the left hand. Four months previous to this admission, the patient stated he had gone to bed feeling quite well, but was awakened during the night with a feeling of numbness in the left hand and forearm. Within an hour the hand and forearm were observed to be quite cold and extremely painful. The pain was of such severity that a physician was called and morphine administered. The following day the pain continued to be severe, and the hand and forearm were observed to be hot. Large doses of morphine were necessary for the relief of the pain. The patient was instructed to bathe the hand with hot water. Discoloration and ulceration of the skin of the fingers developed. The terminal phalanx of the index finger became gangrenous and was amputated. A marked flexion contracture of the fingers developed.

Fig. 1.—Photograph of left hand, November, 1932, four months after onset of contracture. The necrosis and ulceration of fingers probably due to hot applications in the presence of anesthesia.

On examination, the left hand and forearm showed the characteristic appearance of Volkmann's ischemic contracture (Fig. 1). The fingers and thumb were fixed in extreme flexion. The muscles of the forearm were firm. There was complete anesthesia of the entire hand and impairment of sensation in the distal third of the forearm. The distribution of the anesthesia was of the characteristic "glove" type. Pulsations in the radial artery were approximately normal.

The patient was observed for a period of one and one-half months, during which

time the clotting time varied from 6 minutes to 22 hours. Platelet count, 290,000. The blood clot retracted. Blood calcium, 14.89 mg.

Roentgenogram of the hand and forearm showed exostoses of the bones of the forearm and marked atrophy of the bones of the hand and wrist (Fig. 2).

A small incision was made over the flexor muscle of the left forearm under local anesthetic. The muscle appeared pale and there was obviously extensive replacement of muscle fibers by fibrous tissue. A small piece of muscle was excised for examination. Microscopic examination of the specimen showed the characteristic pathologic

FIG. 2.—Roentgenogram of both forearms showing synostosis between left radius and ulna.

changes of Volkmann's ischemic myositis. In some areas the muscle fibers had been completely replaced by dense fibrous tissue. In other areas the remaining muscle fibers were greatly distorted in size and shape (Figs. 3 and 4).

Because of the anesthesia and ulceration of the fingers, it was deemed inadvisable to institute any measures for the correction of the deformity. May 21, 1935, the patient returned for examination. He had almost entirely recovered sensation in the hand. There was normal pulsation in the radial artery. The ulceration of the hand had completely healed. The contracture was unchanged (Fig. 5).

Appended is a brief summary of the reports of the previously recorded cases of Volkmann's ischemic contracture developing in hemophiliacs.

Summary of the Case Reports in the Literature of Five Instances of Volkmann's Ischemic Contracture Developing in Hemophiliacs

CASE I. J. P. Lord:[21] A boy 16 years old, who had hemophilia, developed spontaneous hemorrhages below both elbows, followed by the typical Volkmann's contractures. No biopsy.

CASE II.—Evandro Pasquali:[13] Male, aged eight, with hemophilia. A past history of hemorrhage following trauma. Patient fell and struck his right elbow against the ground, followed in 24 hours by pain in the hand and arm, with a gradual flexion contracture of the hand. No evidence of fracture.

FIG. 3.—Photomicrograph of excised piece of muscle showing complete replacement of muscle fibers by fibrous tissue.

FIG. 4.—Photomicrograph of excised muscle showing an area in which a few abnormal muscle fibers remain in fibrous tissue.

CASE III.—M. Frelich:[22] Boy, four years old, suffering from hemophilia, injured his arm April 11, 1925, followed by marked swelling, and the arm was placed in a sling. In two weeks the swelling disappeared and a typical flexion contracture had begun. No roentgenographic evidence of fracture.

CASE IV.—E. W. Hey Groves:[23] Seven year old male hemophiliac who was pulled by his left arm, in an attempt to extricate him from a crowd. This was followed by bruising, swelling and stiffness of the elbow and wrist, which gradually disappeared, resulting in a wasted, contracted limb.

CASE V.—E. W. Hey Groves:[23] Eleven year old male with hemophilia fell and struck left elbow against the ground. A few days later he noticed the fingers became clawed. A splint was applied for four weeks. After its removal the fingers were found to be in a state of marked contracture.

In a review of the available literature there were found 123 instances in

which Volkmann's ischemic contracture developed without the previous application of any constricting dressing. In 90 of these cases the contracture followed an injury in which a fracture was sustained. The frequent development of ischemic contracture after supracondylar fractures of the humerus in which no constricting dressing is used, 20 per cent according to Steindler,[9] is evidence that this condition is not always the result of defective dressings. Bardenheuer[2] estimates an incidence of 8 per cent of Volkmann's ischemic contracture occurring without previous application of constricting dressings.

In 28 cases a contracture followed an injury in which no fracture had

FIG. 5.—Photograph of both hands and forearms showing extent of the contracture in the left hand, approximately three years after onset.

occurred nor was any constricting bandage applied. The reports of these cases[11-20] are not sufficiently complete to determine in each case the exact severity of the trauma, but one gets the impression that it was moderately severe in each instance. There was no evidence that any of these patients was suffering from an abnormality of the blood. In the six instances of Volkmann's ischemic contracture occurring in hemophiliacs, no constricting bandage had been applied, and relatively little or no trauma had been sustained.

The pathogenesis of spontaneous Volkmann's ischemic contracture in

hemophilia is probably well summarized in J. B. Murphy's statement in discussing an instance in which this condition developed in a patient who sustained an injury of the elbow without a fracture or a constricting dressing. He states: "In our case it is fairly clear that hemorrhage beneath the aponeurosis may cause myositis and subsequent contracture, even when splints or bandages have not been used." Pasquali[10] perhaps summarizes the pathogenicity of spontaneous Volkmann's contracture in hemophiliacs even better when he says in substance: "In hemophiliacs it is comparatively easy for a deep extravasation of blood to occur in the region of the elbow where the particular anatomic disposition of the veins makes the conditions favorable for the development of an ischemia of the nature to produce myositis." In other words, it is not the extravasation of blood into the muscles, but the pressure conditions which result from the extravasations of blood into a space containing muscles, surrounded by comparatively rigid fascia and drained of blood by a peculiar anatomic arrangement of the veins which favors obstruction. It is not improbable that the myositis and subsequent contracture could be averted by a subcutaneous division of the fascia through a small skin incision.

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BRIEF COMMUNICATIONS AND CASE REPORTS

RADICAL EXCISION OF TUBERCULOUS CERVICAL LYMPH NODES AND LOCAL EXCISION OF CARCINOMA OF PAROTID GLAND*

SIX YEAR FOLLOW UP

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CASE REPORT

A. L. (84675), 36 years of age, was admitted to Vanderbilt Clinic, June 13, 1929. At the age of eight he had mumps. At ten he was ill with a pain in his chest, aggravated by breathing, fever, cough, and, at times, hemoptysis. His chest was not tapped, his sputum never examined for tubercle bacilli, and his lungs were not roentgenologically examined. Similar symptoms recurred when he was 17 years old, except that he did not cough blood. At the age of 18, following an accident to the left side of his neck, there appeared a small lump in the middle of the neck just in front of the left sternomastoid muscle. At the end of a year it had increased markedly in size, and it was excised in Lund Hospital in Sweden. Six months following this operation, the swelling reappeared and he was treated by roentgen therapy once every two weeks, for three years, receiving approximately 50 treatments.

Three years later the mass opened and discharged. The sinus was curetted and remained healed for four years. Then, two years after the patient came to America, the swelling reappeared. In 1925, a tonsillectomy was performed at Harper Hospital in Detroit. This did not influence the size of the mass and six months later he was treated, three times a week for five months, with local and general ultraviolet light. During these treatments, the mass reopened and discharged.

When he presented himself at Vanderbilt Clinic he was well developed and well nourished, weighing 148 pounds; temperature, 98.6° F.; urine, negative; red cells, 5,150,000; hemoglobin, 95 per cent; white cell count, 4,800, with 59 per cent polymorphonuclears, 33 per cent small lymphocytes, 3 per cent large lymphocytes, 4 per cent transitionals, and 1 per cent eosinophils. Wassermann, negative. On the left side of the neck was a healed scar, 1 cm. long, 3 cm. below the tip of the mastoid process, to which there was adherent a firm mass approximately 3 cm. in diameter. July 16, 1929, under local anesthesia, this mass was excised.

Pathologic Examination.—The specimen proved to be a mass of cervical lymph nodes 3.5 by 1.5 by 1.5 cm. Microscopic examination revealed active tuberculosis with giant cells and caseation necrosis. There were irregular masses of round epithelial cells with oval pale staining nuclei with mitoses in each second high power field. In another area, the epithelial cells showed an orderly arrangement into glands: *Diagnosis.*—Tuberculosis of cervical lymph nodes, carcinoma metastases to cervical lymph nodes following carcinoma of unknown origin (Figs. 1 and 2).

A search for a carcinoma focus revealed no evidence of malignancy in the nasopharynx, larynx, esophagus or bronchi. August 6, 1929, under colonic ether anesthesia, a radical upper and lower cervical and supraclavicular dissection was done, after the technic of Semken. The operation was followed by a 500 cc. whole blood transfusion.

* Presented before the New York Surgical Society, May 8, 1935.

The pathologic report of the tissue removed showed no tuberculosis or carcinoma in the nodes, but active tuberculosis in the skin and subcutaneous tissue about the previous operative site. June 28, 1934, five years later, the left pre-auricular node became enlarged, and was excised. Microscopically, it showed active tuberculosis.

At the present time—70 months after operation—he is symptomless and without noticeable disability.

Comment.—This case is shown as one of recurrent tuberculosis of the upper superficial cervical lymph nodes which, when excised along with a part of the lower pole of the parotid to which the mass was adherent, showed microscopically a relatively low grade carcinoma in the lower pole of the parotid gland included in the excised specimen. Because of the patient's youth, the presence of carcinoma and tuberculosis in the cervical region, a radical dissection of the upper and lower neck and supraclavicular regions

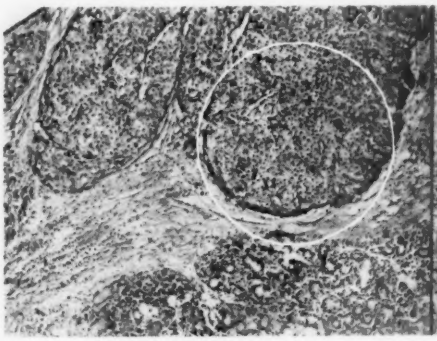

FIG. 1.—Photomicrograph showing carcinoma of the lower pole of the parotid gland.

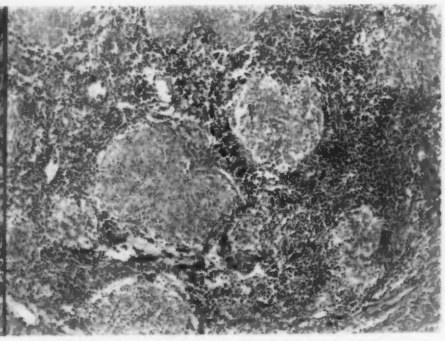

FIG. 2.—Photomicrograph showing tuberculosis of the cervical lymph nodes.

was done, including the excision of the sternomastoid muscle and the spinal accessory nerve in an effort to rid him of both diseases, and, except for a recurrence of tuberculosis in the left pre-auricular node which was excised one year ago, the patient has remained clinically free of evidence of persistence of carcinoma and tuberculosis of the parotid gland and left cervical region for six years.

Rokitansky stated, many years ago, that carcinoma and tuberculosis are antagonistic and rarely occur in the same individual. Since then, however, both have been seen in the same patient many times, particularly in the skin, breasts, stomach, rectum, uterus, cervix, ovary and lungs.

Centanni reported that when tubercle bacilli and tumor tissue were injected in animals, neither grew. Cherry, however, reported the acceleration of spontaneous tumors by the injection of tubercle bacilli. The experimental work is inconclusive.

Pearl, in 1929, from autopsy material at Johns Hopkins, concluded that "a definite and marked incompatibility or antagonism does exist."

Thibaudeau reports from the State Institute for the Study of Malignant Disease, Buffalo, N. Y., that in five years, out of 66,711 specimens examined,

10,897 were carcinoma, and of these 21 had active tuberculosis either in the same area or in regional nodes, as follows:

9 Skin—tuberculosis and carcinoma.

9 Breasts—6 carcinoma and tuberculosis, 3 tuberculosis in the axillary nodes with carcinoma in breast.

1 Cervix—epithelioma with tuberculosis.

1 Uterus (fundus)—malignant leiomyoma with tuberculous endometritis.

1 Stomach—carcinoma metastasis to lung with tuberculosis there.

Of 1,661 breast carcinomata, nine had tuberculosis there also, and in 41 tuberculous breasts there was no carcinoma in the five year period. Of 371 dead of malignancy, 22 showed active tuberculosis at autopsy, or 5.9 per cent with active tuberculosis dying of carcinoma. Two cases of Hodgkins' disease were included as malignant disease. Eleven were squamous cell epithelioma from the mucous membranes. In 15,000 cases of tumors examined for treatment only 22 showed active tuberculosis and cancer. He concludes that associated tuberculosis and carcinoma is uncommon. Apparently there is no antagonism. However, a chronic irritation of a tuberculous lesion may predispose to carcinoma.

Discussion.—Dr. George H. Semken (New York) thought that Doctor Smith's case was unique and that it was surprising not to have had recurrence of cancer follow this limited excision of the parotid gland. Cancer of the parotid is rare. The predominant type of tumor in the parotid is the composite or mixed tumor, which frequently follows the pattern of the fibro-epithelial "cylindroma" (Billroth). These tumors recur when incompletely removed; and some end as malignant new growths with metastases, chiefly in the lungs and bones. Cancer in the region of the parotid may be a primary tumor in the salivary gland, or it may be a metastatic process in the lymph nodes incorporated in the parotid gland.

The radical operation for cancer of the parotid—the removal of the parotid and related cervical lymphatics—is considered one of the most difficult and dangerous in surgery. The deep portion of the parotid extends well into the depth in close proximity to the internal carotid artery, the internal jugular vein, and the vagus nerve; but it is separated from these by a thin fascia which extends upward from the posterior belly of the digastricus. If the dissection is done from below upward and forward, the external carotid artery and the internal jugular vein can be secured early; and, if the fascia from the digastricus is followed as a guide, there will be little likelihood of injury to the great vessels and the accompanying nerves. The branching vessels and the accessory portion of the gland offer no technical difficulties. The facial nerve is necessarily divided; but the resultant paralysis can be overcome in part by a muscle or fascial transplant, or, in suitable cases, by transplanting the lower division or the hypoglossal nerve.

In Doctor Smith's case the interesting question of the significance of the association of cancer and tuberculosis in the same region in one patient was again brought up. This was recently discussed at a meeting of the American Association for Cancer Research; and it was generally agreed that there was no definite relationship between these two conditions, either in a possible predisposition produced by tuberculosis or in an antagonism. In Doctor Semken's experience, many patients with intra-oral cancer, in whom there

had been no cancer metastases in the regional lymph nodes, also had tuberculosis in some of these nodes. This is not surprising when it is remembered that from 25 per cent to 40 per cent of all people (in autopsy records) show evidences of having had some form of tuberculosis; and some of these must certainly come within the group having cancer. One reason why this association is not more frequently noted may lie in the fact that cancer is essentially a disease of older people, while tuberculosis is predominantly a disease of the young. It is probable that this association of cancer and tuberculosis may be dismissed as relatively without significance.

The third question that came up in Doctor Smith's case was that of the treatment of tuberculous lymph nodes. Doctor Semken thought it unwise to emphasize the treatment of such nodes with roentgen rays. This treatment, although seeming to have some influence in healing sinuses from broken down tuberculous nodes, in general has a limited value. There is still a lack of uniformity among radiologists as to the optimal dosage, it being the practice in this country to give frequently repeated small to minimal doses, whereas, abroad, many advise large doses, singly or repeated.

In this disease, the infection is with the human type of tubercle bacillus. The lymph nodes involved are usually those of the carotid packet, regional to the nasopharynx and fauces; and it is generally agreed that the portal of entry has been the tonsils (or possibly nasopharyngeal adenoids). When cervical lymph nodes of other groups seem alone to be affected, it is probable that the invading bacilli passed through the resistant, immediately regional groups, to find lodgement in susceptible nodes farther along the chain. The tubercle bacillus seems to have the power of penetrating the unbroken mucous membrane of the tonsil without leaving any evidence of this invasion; or the small primary ulcers have healed; or there may be a small ulcer in the base of one of the crypts, either not visible or visible only at one edge. It is the usual experience with patients who have tuberculous cervical lymph nodes that no evidence of tuberculosis is found in the tonsils, despite the regional association. Nevertheless, in Doctor Semken's opinion, the treatment of these cases should invariably begin with removal of the tonsils, before any attempt is made to remove the diseased lymph nodes. The failure of early retrogression in the lymph nodes following an adequate tonsillectomy need not be a discouragement, nor does it disprove the casual relationship of the tonsils to the diseased nodes. In one personal experience of this sort, an enforced delay became necessary, in the proposed node dissection following an apparently ineffective tonsillectomy done four weeks earlier. In the ensuing weeks a complete retrogression took place in the relatively large packet of tuberculous lymph nodes, and there has been no reappearance of the disease. The removal of the tonsils and nasopharyngeal adenoids also closes the door to possible later invasions via these portals. The extirpation of the lymph nodes follows the anatomic grouping of these nodes. It may suffice to limit this to the immediately regional group invaded; but the unnoted extensions of the infection often make more comprehensive dissections necessary.

Dr. John M. Hanford (New York) reported the case of an elderly woman in poor condition upon whom he first operated for cancer of the parotid in 1920. The lesion was arrested for some time, but finally reappeared, and in 1930 she was again operated upon, after radium had been applied. The swelling subsided and did not reappear until a few months ago when it occurred in the anterior part of the parotid and reached a size of 3 or 3.5 cm. in diameter. It was very hard, and definitely malignant. Thus for

15 years, on and off, this patient has had—and still has—carcinoma of the parotid gland. There has been no doubt about the diagnosis. She is fairly well and has never had metastases to the lymph nodes. For many years, however, it was considered that she had very definite and quite extensive metastases to the lung. But she continued well, except for the occasional local recurrences. The round shadows in the lungs that we thought typical of malignant metastases remained stationary. On her last visit to the hospital the roentgen ray department concluded that the findings in the lungs were not metastases but a very peculiar and unusual type of tuberculous masses. In any event the lung findings have been present for seven or eight years, without change.

With regard to tuberculous nodes, Doctor Hanford strongly disagreed with the practice of taking out the tonsils first. He thought that they were rarely the primary seat of infection and pointed out that tubercle bacilli may go through the tonsils without causing a primary lesion and that it is very rare in most people with tuberculous cervical lymph nodes to have tuberculosis of the tonsils. Even when they do, the disease in the lymph nodes is already established; the process is under way; and removal of the tonsils is not more likely to cause an arrest of the tuberculous process in the neck than is removal of one of the lymph nodes. In his opinion it is much better to treat the lymph nodes first, especially if one plans to remove them. Liquefaction may go on in a very short time. Tonsillectomy predisposes to liquefaction. Hence, it is much better to operate on the nodes before this occurs.

Dr. John E. Jennings (Brooklyn) expressed his general support of Doctor Semken's attitude and mentioned the decrease in recent years of tuberculous nodes of the neck. Some 20 or 30 years ago there were regularly five to ten or more cases in the wards, whereas he had not seen a case of this kind in recent years. He said that this is accredited to the removal of the tonsils, and by public health men as the result of better milk. Whatever the cause, in his experience tuberculous nodes in older children and young adolescents had almost disappeared.

Dr. F. W. Bancroft (New York) stated that at Fifth Avenue Hospital, which is at present a drawing area for Porto Ricans and Central Americans, there are a good many cases of tuberculous cervical adenitis, there being two or three there at the present time, as well as one woman with a large tuberculous adenitis of the retroperitoneal lymph nodes.

Dr. Charles E. Farr (New York) cited two cases in which he had performed a radical operation for carcinoma of the parotid. In both cases, he was able to preserve the facial nerve. In one patient, a comparatively young woman, the condition was recurrent when he first saw it. The operation plus roentgen therapy has kept her alive and well four or five years. The other patient, an older man with a more extensive carcinoma, stayed well for two years after a radical operation, then disappeared. He doubted that either was a permanent cure.

Dr. Herbert Willy Meyer reported a case of adenocarcinoma of the parotid operated upon by him at the Lenox Hill Hospital. The patient had a small lesion near the lower pole of the parotid, considered a mixed tumor. Excision was done and it proved to be a very active carcinoma. Three days after the primary operation a complete radical operation was done, the entire parotid gland being removed, together with a block dissection of the

cervical lymph nodes. Metastases in the lymph nodes of the lower chain were found. The patient had, of course, paralysis of the facial nerve. Plastic repair of the eye and angle of the mouth with two strips of fascia lata was done to overcome the paralysis. The patient is now over 70 years of age, is happy in spite of the facial paralysis, does not look very much deformed, and has been well for six years.

Dr. Frank E. Adair pointed out the great diagnostic help furnished by the use of a sialogram injection. After drawing out a little fluid, the duct is injected and roentgenograms made which reveal a good deal about the pathology of the parotid gland: particularly whether there is a stone obstructing it, whether there is an inflammatory condition of the gland, or whether there is a carcinoma. He thought that the cure in the case under discussion might be partially attributable to the pathology. Ordinarily, carcinoma of the parotid cannot be cured by such a simple surgical extirpation as that carried out by Doctor Smith. From a study of Fig. 1, the lesion does not seem to be a very active type of carcinoma, in that it is not infiltrating and not completely circumscribed. Even though it has mytotic figures, some pathologists would not call it carcinoma without infiltration.

Doctor Smith explained that the tissue removed at the radical operation showed no carcinoma in the portion of the parotid then removed; at the second operation, there was none in the cervical lymph nodes but the nodes showed tuberculosis. Dr. A. P. Stout diagnosed the lesion as a small, low grade carcinoma, there being infiltration in the gland itself and in the capsule, apparent in parts of other sections.

CONGENITAL ATRESIA OF DUODENUM IN NEWBORN*

DUODENOJEJUNOSTOMY

Edward J. Donovan, M.D.

New York

Case I.—Baby F., a cesarean delivery, was transferred from Sloane Hospital to Babies Hospital, with a history of having vomited bile stained material since birth. The mother had one other living child and one labor was terminated by craniotomy.

The patient weighed seven and one-half pounds at birth, but lost about one-third of this in the next eight days. She vomited continuously following birth, and her stools, said to have been passed daily, consisted largely of meconium. Becoming dehydrated, she was kept alive by clyses, etc. Roentgenograms on the ninth day of age disclosed obstruction in the third part of the duodenum.

The abdomen was opened through a right subcostal incision. The first and second portions of the duodenum were almost as large as the stomach. This dilatation seemed to extend downward to the point where the superior mesenteric vessels passed over the duodenum. The jejunum beyond the duodenojejunal junction was completely collapsed. There was no other demonstrable cause for the obstruction. The jejunum about 10 cm. beyond the junction was brought anterior to the colon and anastomosed to the lower part of the dilated duodenum by three rows of sutures. Continuous intravenous drip of glucose in saline was started and continued for 60 hours. Convalescence was uneventful except for some postoperative vomiting for the first five days. After the fifth day, there was no vomiting and the child made rapid progress.

* Presented before the New York Surgical Society, May 8, 1935.

It is now seven months since operation. The child does not vomit and is gaining weight normally, now weighing 16½ pounds. A roentgenogram shows a normal duodenal cap and that the duodenum is almost completely empty within 20 minutes.

CASE II.—C. B., a male child, was admitted to Babies Hospital at the age of 17 days, with a history of having vomited everything since birth. He was an instrumental delivery. He was taken off his mother's milk and put on SMA, but still vomited after every feeding, and in this period lost almost two pounds. The vomiting was not projectile, but was always bile stained. The history was that the stools had always been of normal color.

Physical examination revealed a large child showing considerable loss of weight. Otherwise it was negative except for the upper abdomen, which was distended, with visible peristaltic waves passing from left to right. Roentgen examination showed a duodenum that was very large in its first and second portions. Small amounts of barium passed through the dilated duodenum at 40 minutes.

OPERATION.—The abdomen was opened through a right subcostal incision and the stomach, except for some dilatation, was found to be normal. The first three parts of the duodenum were as large and thick walled as the stomach. The jejunum beyond the duodenojejunal junction was about half normal size. The obstruction was at the point where the superior mesenteric vessels passed across the duodenum. An anterior duodenojejunostomy was done by suture, a point on the jejunum where it could be approximated without tension being selected for anastomosis. Three rows of sutures were used, leaving a stoma about one inch long, running transversely. Following operation fluids were administered intravenously.

The child vomited bile stained fluid several times during the first five postoperative days. The stomach was emptied by a Levine tube. From the fifth day on convalescence was uneventful except for a subacute otitis. On February 16, 20 days postoperative, he was discharged.

Three months have elapsed since operation. The child now weighs 16½ pounds and does not vomit. His incision has healed soundly. Roentgenograms during the past week show a normal passage of barium through the stoma in 20 minutes.

DISCUSSION.—DR. CHARLES E. FARR (New York) called attention to the remarkable fact that most surgery in the abdomen in infants is in the right upper quadrant. Referring in particular to the two cases upon whom a duodenojejunostomy was done, he regarded the incision used by Doctor Donovan as a very useful one for the exposure needed. In only occasional cases is any other incision necessary, and in these one is quite aware of that necessity before operating. Although Doctor Farr had not had a case of complete atresia of the duodenum necessitating a duodenojejunostomy, he had had quite a number of cases of fairly complete atresia or stenosis requiring surgery, occurring months or years after birth. The etiologic factor was usually found to be due to bands, kinks, or misplacements of viscera, not requiring a duodenojejunostomy. He felt that Doctor Donovan was especially to be congratulated on his marvelous skill in handling very little infants with their extremely delicate tissues. The greatest operative difficulty, he emphasized, is not with the dilated portion of the duodenum, but with that part which has never functioned, or, if it has, only very slightly. Even when successful in making a competent anastomosis one cannot be sure that the portion that has never functioned is going to take up its work and carry on, as exemplified in Doctor Farr's experience, by disastrous results in the lower bowel where, at postmortem, it was apparent that technically the work was good but obviously function had not occurred. Doctor Stockard's suggestion is to start at once with enemata and give them rather fre-

quently so that the bowel may be forcefully drained from below and peristalsis stimulated.

Doctor Donovan stated that he had performed an antecolic duodenojejunostomy rather than a retrocolic duodenojejunostomy in these cases because the transverse mesocolon in a baby so young is very short and very thin. By doing an antecolic operation, the danger of interfering with the middle colic artery is lessened. The technic is the same as that of an anterior gastro-enterostomy, the jejunum being anastomosed to the dilated duodenum with three rows of sutures and the stoma running transversely. The only objection to the method is the possibility of a little difficulty in making the stoma low enough on the dilated duodenum to drain it properly, but in all of Doctor Donovan's cases this has worked well. An entero-enterostomy was not necessary in any of the five cases in his experience.

PSEUDOMYXOMA PERITONEI*

PROBABLY OF APPENDICEAL ORIGIN

Frank L. Meleney, M.D.

New York

In February, 1933, this patient was first presented to the New York Surgical Society.[1] He was a man of 45 years with a tender mass in the lower abdomen extending somewhat more to the left than to the right of the midline. It proved to be a mass of infected tumor tissue containing quantities of mucus and a disorderly but well differentiated growth of mucous membrane. The consensus of opinion was that it had probably arisen from an appendix which had extended across the midline to the left

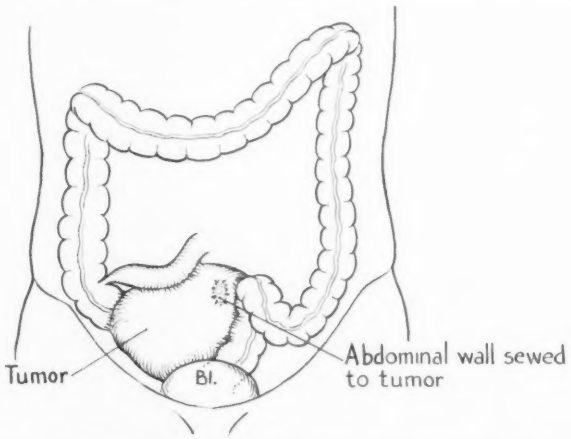

Fig. 1.—First operation. Showing what was inferred to be its general anatomic relations.

side and had probably ruptured and transplanted some of its mucous membrane in the peritoneal cavity (Fig. 1). It seemed to have subsequently invaded the lower ileum and the sigmoid for fecal fistulae had been established at all three of these sites. By a series of operations, including an amputation of the cecum, an ileo-colostomy with a tape ligature of the ileum below the tumor, and a permanent left inguinal descending colostomy,

* Presented before the New York Surgical Society, April 24, 1935.
[1] Annals of Surgery, vol. 98, pp. 458-460, 1933.

the tumor was shut off from the fecal stream (Figs. 2 and 3), but it was still present in the abdomen, the mucous membrane was producing large quantities of mucus and the tumor seemed to be invading the abdominal wall. At that juncture, the case was presented with the hope of obtaining some advice concerning further procedure to eradicate the disease. No suggestions were offered.

In March, 1933, the tumor continuing to pour out purulent mucus, an attempt was made to remove it from within the cavity of the tumor, but in doing so the peritoneum was opened. The stump of the ileum and the stump of the sigmoid were brought up into the wound and the skin sutured to the outer wall of the tumor as far posteriorly as possible (Fig. 4), about one-third of the tumor was removed. The pathologist (Dr. A. P. Stout) then diagnosed it colloid carcinoma. No peritoneal infection developed and two weeks later another attempt was made to remove the tumor from within its own cavity. The mucous membrane was shelled out from the wall of the cecum and from the ileum down to the tape ligature near the ileocecal valve. Likewise the mucous membrane was shelled out of the sigmoid as low down as possible and cut across. The lower cuff was then inverted and the muscular wall of the sigmoid approximated over the inverted mucosa. The tumor was then gently curetted off the posterior abdominal wall. Below the mesentery of the ileum it seemed to have invaded the deeper tissues. Again the pathologist made a diagnosis of colloid carcinoma. In view of this fact we decided to apply radium to the remaining cavity. Dr. Maurice Lenz gave a maximal dose ten days after the excision, 150 mg. being applied for a total of 3,450 mgh. The cavity then began to contract down. One month later, the tape previously used to tie off the ileum sloughed out. The cavity then contracted to about 1 cm. in diameter and 3 cm. deep, and continued in this relatively innocuous state for eight months. Then there appeared, in the lower wall, a granular area which began to secrete mucus. The cavity began to grow larger. An attempt was made to excise the whole area of the cavity. In doing so the peritoneum was entered but again healed without infection. The specimen again revealed disorderly epithelial tissue which was called carcinoma.

With the patient completely aware of the situation we proposed an attempt at radical removal. March 10, 1934, the peritoneum was frankly entered and dissection carried out from above downward. We were surprised to find how cleanly it could be removed from the posterior wall of the abdomen and pelvis. The rectum was cut across low down and the stump inverted (Fig. 5). A large area of anterior abdominal wall was removed. The closure was difficult. The bladder was brought up to close the defect in the peritoneum. This was covered by a large piece of fascia lata. The patient was kept bent forward by means of the Gatch bed and the wound healed with surprising firmness. The patient left the hospital on the twenty-third day and gained steadily in strength. The large piece of excised tissue was carefully searched for tumor but no trace could be found. He returned to work a month later and has steadily improved. The colostomy functions satisfactorily although there is a weakness of the abdominal wall around it. He has gained 100 pounds in the last year and apparently has recovered completely.

Dr. A. P. Stout (New York) explained that, in regard to the origin of the epithelium, it had never been possible to determine where it came from, but that the most probable source in pseudomyxoma of the peritoneum in the male is the appendix. It was hoped during the course of the various operations that the appendix would be seen so that it might be determined whether it came from that, but it never was. Another possibility was that it might have come from an enterocystoma, although that is a much rarer lesion. These cases are really extraordinary because the epithelium of the mucous membrane of the gut behaves as if it were malignant tissue, growing out over the surface very persistently, and yet it usually does not infiltrate or become cancerous. There was a time during the course of these operations

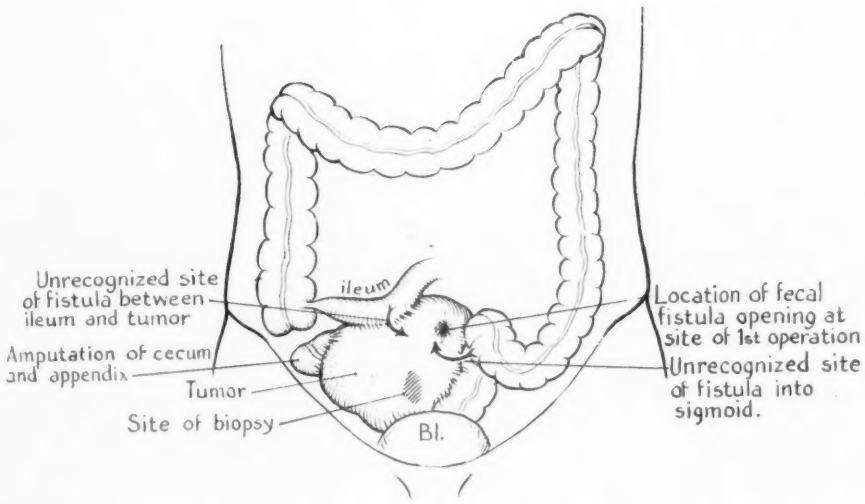
Unrecognized site of fistula between ileum and tumor
Amputation of cecum and appendix
Tumor
Site of biopsy
ileum
Bl.
Location of fecal fistula opening at site of 1st operation
Unrecognized site of fistula into sigmoid.

FIG. 2.—Fourth operation. An effort made to cut off the fecal flow from the tumor by amputation of the cecum.

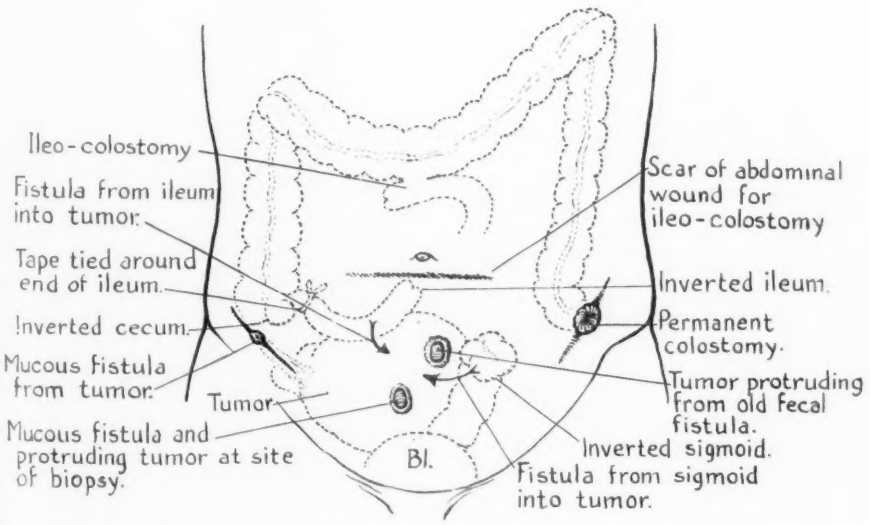

FIG. 3.—Fifth operation. Fecal flow cut off from tumor by permanent colostomy, ileo-colostomy and ligation of ileum near the ileocecal valve.

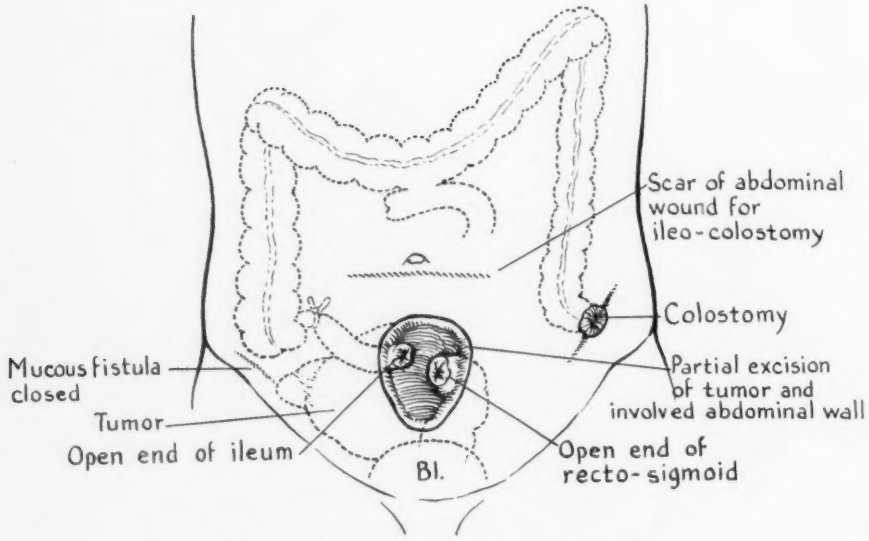

FIG. 4.—Sixth operation. Partial removal of tumor.

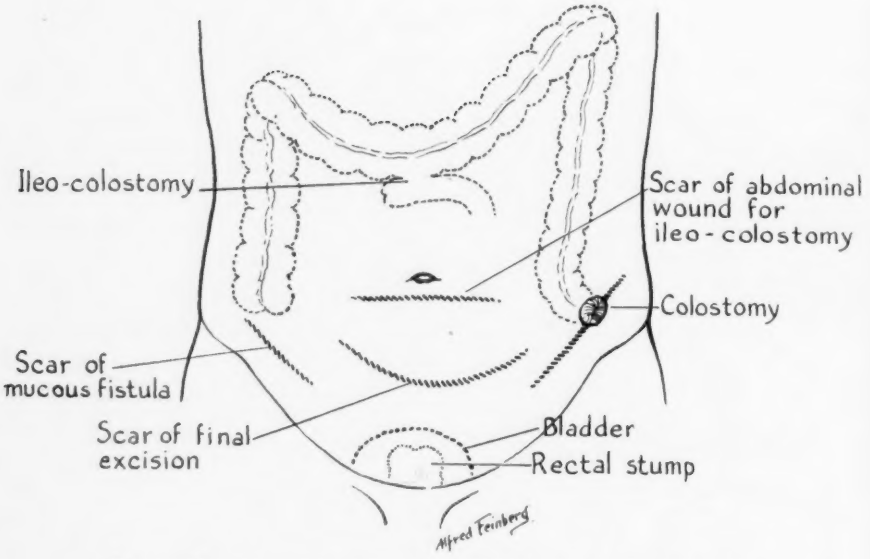

FIG. 5.—Complete removal of tumor. Showing the final anatomic relations.

when it seemed as if a cancerous change had occurred; that is, as if the epithelium had invaded the underlying tissues. There was so much mucin that some of the epithelial cells were actually floating around in it just as they do in colloid carcinoma. This led to the supposition at one time that it had become cancerous, but the outcome seems to have proved that this was not so and that the case is actually one of pseudomyxoma of the peritoneum.

RUPTURE OF THE LIVER

FREDERICK CHRISTOPHER, M.D.
EVANSTON, ILL.

THE mortality in unoperated cases of rupture of the liver is probably about 80 per cent[1] and in operated cases about 60 per cent.[2] Reports of ruptures of the liver are not common, although it is regarded by some as the most frequently injured internal organ.[3] Ruptures of the liver have been classified as: (1) rupture of the hepatic tissue and capsule; (2) rupture of the hepatic tissue without rupture of the capsule (subcapsular rupture); and (3) central rupture of the hepatic tissue.[1] Although in subcapsular ruptures, if they can be identified, operation is somewhat less urgent than in those cases where the capsule is torn in addition to the hepatic tissue, for general purposes it is probably wise to follow the advice of Moynihan,[4] who believes that in suspected cases the need for operation is instant. Robertson and Graham[5] report two cases of subcapsular ruptures of the liver and cite three others from the literature. In both of their cases the diagnosis was based chiefly upon the large epigastric tumor. The first case was successfully operated upon some 30 hours after the injury. The second case, in which the tumor did not appear until over two weeks after the injury, was not operated upon until 27 days after the injury when gross destruction of the liver tissue was found.

The following case presents several unusual features. First, there was a primary subcapsular rupture of the liver followed in less than 24 hours by a spontaneous rupture of the capsule on the operating table. Second, the subphrenic space became infected by a hemolytic Streptococcus requiring rib resection for drainage. Third, during the convalescence, the patient twice developed acute intestinal obstruction requiring laparotomies with prophylactic enterostomies.

CASE REPORT

K. N., aged 12, was admitted to the Evanston Hospital June 10, 1935, at 11:30 P.M. Three hours previously, while climbing a blunt wooden picket fence, she had fallen, striking her chest heavily against one of the pickets. A small abrasion just to the right of the xiphoid process marked the point of impact. When seen very shortly afterward she was suffering a great deal of upper abdominal pain, pulse 170, and was in considerable shock.

On admission to the hospital, the pulse was 100; temperature, 98° F.; and blood

pressure 135/70. The leukocyte count was 19,000; erythrocytes, 3,700,000; hemoglobin, 65 per cent. There was extreme tenderness and some rigidity of the upper right quadrant of the abdomen. Roentgenologic examination of the chest failed to disclose fluid and that of the abdomen in the vertical position gave no evidence of a pneumoperitoneum.

During the night the pulse rose steadily and at 8 A.M. was 150, weak and irregular. A tender mass, apparently liver, protruded four fingerbreadths below the costal border. At 9 A.M. the blood pressure was only 87/52 notwithstanding a hypodermoclysis of dextrose Ringer's solution which was in progress. Gum acacia solution, 1,200 cc., was given intravenously and by 11:30 A.M. the pulse was 130 and of good quality. A citrated blood transfusion (325 cc.) was then given. At 12:30 the pulse was 110 and of good quality, the hemoglobin 44 per cent and the erythrocytes 2,090,000. The upper right quadrant now presented a marked rounded, tender swelling. The pulse having increased to 146 by 4 P.M., an exploratory laparotomy for rupture of the liver was determined upon.

FIG. 1.—First intestinal obstruction. Note distended loops of small bowel.

Just before making the abdominal incision at 5:45 P.M. (June 11, 1935) it was noticed that the rounded swelling in the upper right quadrant had disappeared. On opening the peritoneum there was a gush of bright red blood. This was found to have come from a subcapsular hematoma which had involved practically the entire upper surface of the right lobe of the liver, and which probably had ruptured spontaneously after the anesthesia (nitrous oxide-ether) had been begun.

The right rectus incision was enlarged by cutting transversely across the right rectus muscle. From beneath the torn liver capsule handfuls of clots and large quantities of fluid blood were evacuated. The ragged surface of the liver parenchyma was felt over the entire upper surface of the right lobe up under the diaphragm. In the hurried palpation, no distinct fissure was recognized. The raw, bleeding surface of the liver was tightly packed with four large rolls of vaginal gauze packing. A 500 cc. blood transfusion was given during the operation. At its conclusion the pulse was 156, blood pressure 110/40, general condition satisfactory.

On the day following the operation the pulse rose to 166, temperature to 103.4° F. Blood and gum acacia were given intravenously and 5 per cent dextrose Ringer's solution was given subcutaneously. On the third postoperative day the pulse dropped from 160 to 120.

On the eighth postoperative day the gauze packing was removed and cigarette drains inserted. These were subsequently shortened and gradually removed. The patient improved rapidly and was sitting up on the twelfth postoperative day. On the fifteenth day, the patient began to have cramp-like abdominal pain and vomiting, which became worse the following day. By the seventeenth day the condition was much worse. The pain was more severe and the vomitus became dark brown. Roentgenologic examination of the abdomen (Fig. 1) showed a typical small bowel obstruction. A laparotomy for intestinal obstruction was performed. On opening the peritoneum a large quantity of thin blood-tinged fluid escaped. The small bowel loops which presented were markedly

FIG. 2.—Second intestinal obstruction. Distended loops of small bowel are lower in the pelvis than in the first obstruction. The stomach contains gas.

distended and of reddish color. The obstruction was found high up on the right side near the ascending colon and was caused by a plastic adhesion. This was divided and a mushroom catheter enterostomy performed about eight inches proximal to the site of the obstruction. A nasal catheter connected to the Wangensteen suction apparatus was inserted into the stomach. On the twenty-second day (three days after the second operation) the patient became listless and the pulse and temperature increased. Some days previously the last of the cigarette drains had been removed and it was feared that a subphrenic infection and abscess formation might be taking place. The roentgenologist made an examination with the patient lying on her side, and found undue elevation of the right diaphragm and an air bubble in the right subphrenic region which suggested subphrenic infection or abscess due to an aerogenous organism.

On July 2, 1935 (the twenty-third day after the first operation), a third operation was undertaken. An incision was made over the tenth right rib in the anterior axillary line. Beginning at the costochondral junction an inch and one-half of rib was resected.

The visceral and parietal pleura were sutured and after positive needle aspiration an incision was made through the diaphragm. A cavity some 6 by 4 by 1 inches was found containing gray clots and old blood. Cultures showed a long chain Streptococcus. On the twenty-sixth day the bowels were functioning and the enterostomy tube was cut off, leaving the mushroom portion inside of the bowel to be passed.

On the twenty-seventh day the patient again began to have cramp-like abdominal pain and vomited. Roentgenologic examination (Fig. 2) again suggested small bowel obstruction. A blood transfusion of 500 cc. was administered and the fourth operation was performed (July 6, 1935). The distended bowel had a dark color and the obstruction was found to be on the right side, lower in the abdomen than the previous one but some 20 inches proximal to it on the small bowel. The obstruction, which was due to an adhesion, was corrected and a mushroom catheter enterostomy performed. Wangensteen nasal catheter suction was again instituted and continued for about 36 hours.

From this time on, convalescence was without serious incident. The thoracotomy cavity was irrigated until it finally closed. The second laparotomy wound became infected and the skin had to be incised on the thirty-seventh day (July 16, 1935) at which time the second enterostomy tube was removed. The child gradually improved and took a few steps on the fifty-eighth day. She was discharged from the hospital on the sixty-sixth day; the wounds healed entirely soon after. She gained rapidly in weight, and apparently is in perfect health.

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PATHOLOGIC FRACTURE THROUGH MYELOMA OF SHAFT OF FEMUR*

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NEW YORK

CASE REPORT

THE symptoms of this patient (No. 223,302), a white male, 45 years of age, married, began August 6, 1929, with a slight pain in the upper left femoral region which affected him only when he was on his feet and physically active. In the early part of September, 1929, while doing strenuous work, he was seized with a "mean" pain in the same region, so severe that he had to sit down. Following this he was treated symptomatically for a time but did not improve. A roentgenologic examination made several days later revealed a tumor in the upper left femur (Fig. 3A).

After a number of consultations he was operated upon October 14, 1929, by Doctor Bloodgood. At operation an extensive central lesion of the upper shaft of the femur was found. The tumor, described as a very bloody sponge-like mass, was curetted and cauterized. It proved to be a myeloma of the plastma cell type (Fig. 1).

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* This case was originally reported by Dr. Charles F. Geschickter: Multiple Myeloma as a Single Lesion. ANNALS OF SURGERY, vol. 92, pp. 425–533, September, 1930. The present follow up report was read before the New York Surgical Society, April 24, 1935.

During convalescence very little new bone formation appeared and the patient suffered a pathologic fracture. Treatments with Coley's toxins were then begun and according to Doctor Bloodgood's report pain ceased almost immediately after the institution of this treatment. The formation of new bone was observed soon afterward. The toxins were continued over a period of about three weeks and were then stopped because the patient had a severe convulsion following an injection and refused further treatment. February 28, 1930, about four and one-half months after operation, the patient was permitted to leave the hospital on crutches. He was thought to have fairly good union at that time and returned to New York March 1, 1930. The next morning, while getting out of bed, there was a sharp, sudden "snap" in the upper left thigh with excruciating pain. He remained in bed and made no further attempt to move the leg.

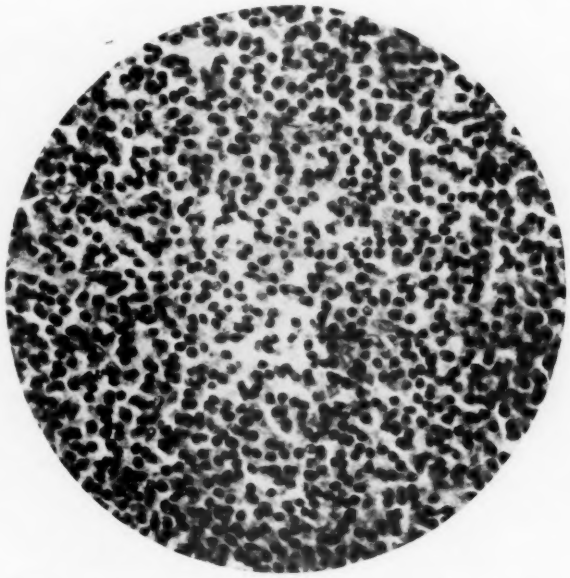

FIG. 1.—Photomicrograph of tumor. Myeloma of plasma cell type.

Examination revealed external rotation and about 4 cm. shortening of the left leg. There was a long scar over the lateral surface of the upper shaft of the femur with two sinuses which discharged thin, seropurulent material. At this level there was considerable external bowing of the femur. The patient appeared to be in great pain and there was marked tenderness.

A Thomas splint was applied and moderate traction maintained by the use of moleskin adhesive straps. The patient was kept in bed at home for a few days until the acute pain and tenderness had subsided. On March 10, 1930, he was transferred to St. Luke's Hospital where roentgenograms showed a fracture of the left femur through what appeared to be a new growth just below the lesser trochanter (Fig. 3B).

Under the direction of Dr. F. C. Wood a series of 11 deep roentgen ray treatments were given between March 11 and June 30, 1930. Relatively small doses, 300 r, were given at weekly intervals. At the end of this period of about 15 weeks, roentgenologic examinations of the fracture showed union of the fragments. The patient was then fitted with a caliper type of brace and allowed to get about on crutches. He was discharged from the hospital July 7, 1930, about four months after admission. Roentgen ray therapy over the affected area has been repeated at intervals up to the present time.

No further foci of the disease were discovered until October, 1932. At that time

the patient complained of pain in the lumbar region and a radiogram showed some destruction of the body of the third lumbar vertebra (Fig. 2A). Under deep roentgen ray therapy the pain disappeared and the lesion apparently healed. A roentgenologic study of the patient's skeleton made October, 1934 (Figs. 2B and 3C), revealed no suspicious areas except a rarefaction of the head of the right fibula.

The urine generally has been free of Bence-Jones protein. In November, 1934, however, the patient complained of thirst and polyuria and a urine examination made at that time revealed a large amount of the Bence-Jones protein.

The patient now has firm union with about 6 cm. of shortening. He was able to discard his brace and now, three years after the fracture, walks, usually with a cane, but sometimes without it. He has shown, however, a progressive anemia of the sec-

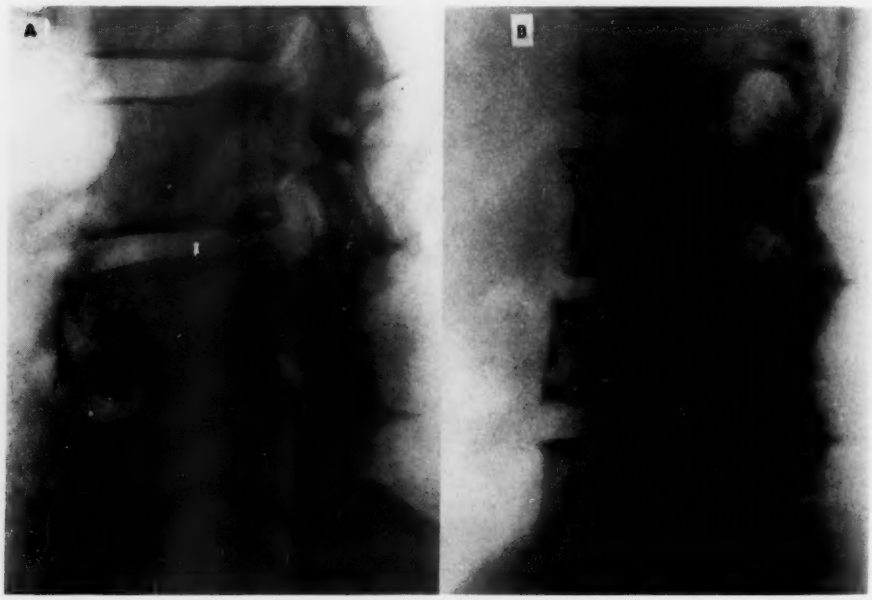

FIG. 2.—(A) October 25, 1932, destructive lesion in body of third lumbar vertebra. (B) February 1, 1934, appearance following roentgen ray therapy.

ondary type, the last count being: red blood cells, 1,700,000; hemoglobin, 31 per cent; white blood cells, 6,000; polymorphonuclear leukocytes, 64 cent cent; large monocytes, 36 per cent.

Several remarkable features of this case deserve emphasis. First, the disease had at its onset and for a considerable length of its course but a solitary demonstrable focus of bone involvement. Second, both the initial and secondary lesions showed response and healing under irradiation despite a pathologic fracture. Third, the appearance of the secondary or new foci of the disease in the lumbar spine did not occur until three years after the initial lesion in the femur, and Bence-Jones bodies were not demonstrated in the urine until five years after the clinical onset.

DISCUSSION.—DR. WILLIAM B. COLEY (New York) called attention to a paper on multiple myeloma[1] in which he described 15 cases treated under his

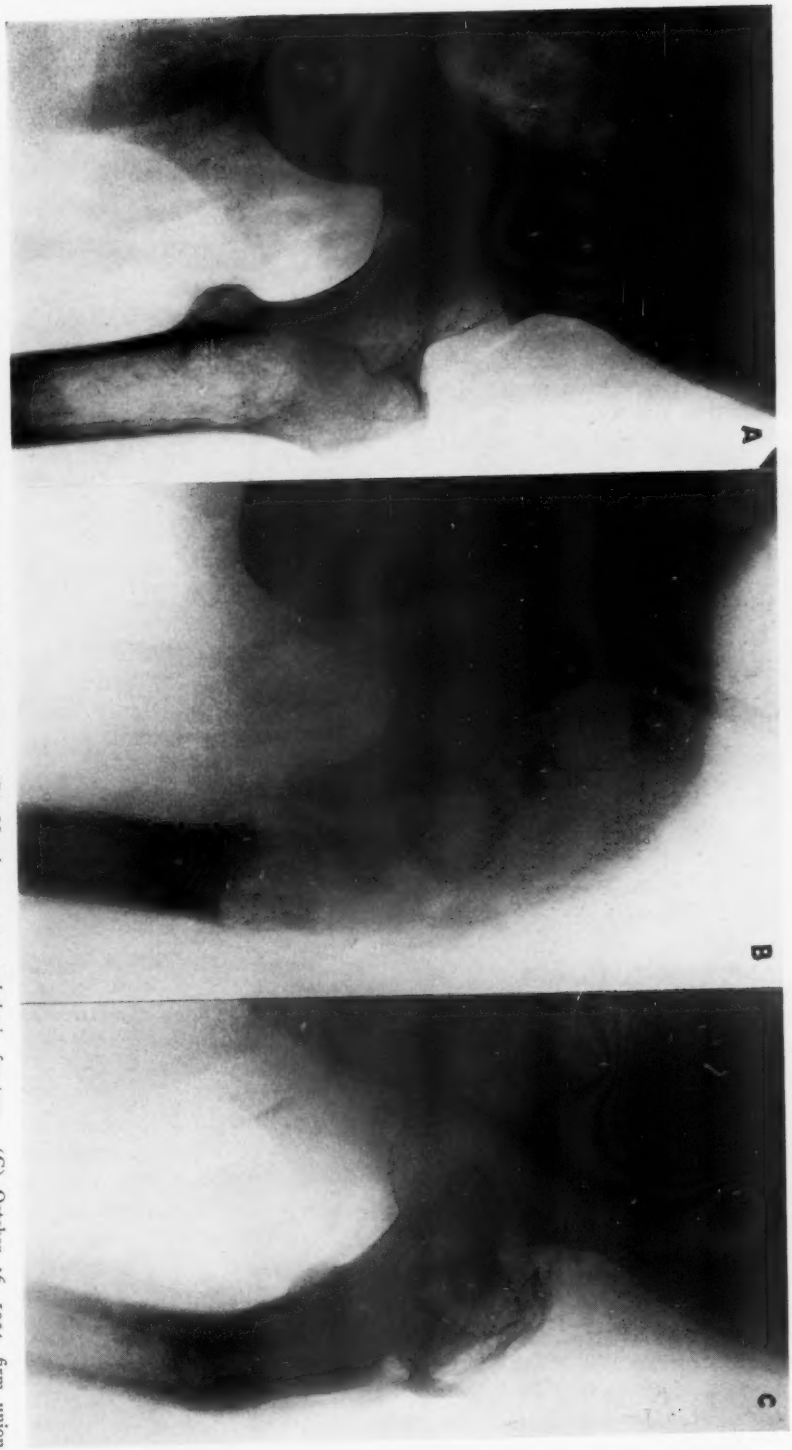

FIG. 3.—(A) September 14, 1929, the original lesion in upper shaft of left femur. (B) March 2, 1930, pathologic fracture. (C) October 16, 1934, firm union following treatment.

direction (including that just presented by Doctor MacFee) and quoted therefrom as follows: "That multiple myeloma is always fatal, and that no treatment is known of any value even in checking the progress of the disease, has been so universally accepted that the question of treatment is scarcely referred to in the very considerable literature on the subject. Geschickter and Copeland,[2] in a recent and most valuable monograph covering some 56 pages, devoted only six lines to treatment. They state that with no proved case reported as cured it is evident that palliative, symptomatic treatment only is available."

These two authors, according to Doctor Coley, overlooked the case of Dr. J. H. Thomas,[3] in which an operation for a tumor of the dorsal spine had been performed, and a microscopic diagnosis of typical multiple myeloma made. The patient later developed metastases to the ribs and partial paraplegia. He was then under the care of Dr. Nathaniel Hunting of Quincy, Mass., who wrote to Doctor Coley seeking advice on the use of toxins. The condition was regarded as sufficiently analogous to round cell sarcoma to justify trying toxins, and the patient showed almost immediate improvement, all evidence of the tumors, as well as the paraplegia, gradually disappearing. The toxins were kept up, once or twice a week, for two years. The patient remained well for five years and then died of lobar pneumonia. There was no evidence of any recurrence of his former trouble at the time of his death.

In addition to the 15 cases reported in 1931, four additional cases of multiple myeloma had since been treated. While in the majority of cases the treatment (toxins alone or toxins combined with irradiation) has had only palliative or temporary effect, the results in these instances have been striking.

Case Report.—L. L. A., a male, 56 years of age, had complained of pains in the lumbar and abdominal regions in 1923, for which he was given high-voltage roentgen therapy. In May, 1924, he was operated upon by Doctor deMartel of Paris for a malignant tumor of the dorsal spine. Extensive postoperative irradiation was given. He remained well until the early part of 1928, when he had a local recurrence with paraplegia and involvement of several ribs. He consulted Doctor Regaud of Paris, who did a biopsy in March, 1928. Sections of the tumor of the ribs were examined by leading pathologists of Paris and pronounced multiple myeloma. Doctor Regaud said that nothing more could be accomplished by irradiation and urged the patient to try Coley's toxins. Irradiation was continued but the condition grew steadily worse. The patient returned to America and in August, 1928, his local physician, Dr. Thompson Anderson, of Denver, Colo., consulted Doctor Coley with regard to using toxins. In view of the patient's condition (complete paralysis of the bladder, rectum and lower extremities, paralysis of one vocal cord, loss of 50 pounds in weight, and large bed sores), Doctor Coley did not encourage the use of toxins, believing that the disease was too far advanced to justify hope for improvement. Nevertheless, the patient insisted on a trial and toxin treatment was begun by Doctor Anderson under Doctor Coley's direction. Immediate and rapid improvement followed. In a few weeks, the patient was sitting up, and in a few months was able to get up. Roentgenograms of all the skeletal bones in September, 1929, failed to show any evidence of lesions. The patient had regained his normal weight and had returned to his work. He remained in good condition until the latter part of 1932, more than four years later, when his symptoms returned, and he died in January, 1933.

In September, 1929, Doctor Bloodgood learned of the result obtained in the foregoing case and asked Doctor Coley to send him some toxins for use in a case of solitary plasma cell myeloma of the femur with pathologic fracture, *viz.*, the case just presented by Doctor MacFee.

Although it would be impossible to estimate the part played by the toxins in controlling the disease in this patient over a period of more than five years, in view of the very small doses given (much smaller than advised) and the much too short term of treatment, Doctor Coley felt inclined to attribute the larger share of credit for the successful result to the roentgen therapy. Nevertheless, the only two cases reported of multiple myeloma that have remained well for a period of five years or more received Coley's toxins, one toxins alone, the other in conjunction with irradiation. A case of plasma cell myeloma more recently treated with toxins and roentgen therapy is well, two and one-half years later.

CASE REPORT.—P. McC., male, 39 years of age, caught a severe cold in June, 1932, and suffered pain all over his body. The cold cleared up, but not the pain. In the latter part of September, 1932, he was admitted to Bellevue Hospital, on the service of Dr. Alexander Lambert, where a diagnosis of multiple myeloma was made. Doctor Coley saw the patient in consultation and advised toxins in conjunction with roentgen therapy. The patient was transferred to Memorial Hospital November 14, 1932. Roentgenograms showed areas of bone destruction in the spine, ribs, skull and pelvis, together with bone alteration in the femora. The toxins started at Bellevue Hospital were resumed and the patient proved very susceptible. On December 14, he was placed in the Heublein unit where he remained for a period of two weeks, receiving continuous roentgen therapy all over his body. By January 29, 1933, he was feeling so well that he insisted on returning to his home in Ireland. When last heard from on January 14, 1935, he was in excellent health without evidence of recurrence.

In recent years three cases of solitary multiple myeloma have been treated by primary irradiation at Memorial Hospital:

CASE REPORTS

CASE I.—J. C., male, 48 years of age, had a solitary myeloma of the humerus. This was treated with radium pack in October, 1929. The patient developed a pathologic fracture in January, 1930, and died a few months later.

CASE II.—A man with a solitary myeloma of the femur of two or three year's duration was admitted in August, 1926. He was treated by Doctor Herendeen with irradiation, the lesion being held under control for three years, after which it grew worse and the patient died.

CASE III.—A patient with a like lesion of the upper femur that was held in check for two years, when it involved other skeletal bones.

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Annals of Surgery
March, 1936

AN IMPROVED INTRAVENOUS ADAPTER

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AMONG all the unwelcomed accidents of a minor nature which may occur during blood transfusion or other intravenous therapy, perhaps none is more disconcerting and embarrassing to the operator than to have the needle unexpectedly leave the vein before completion of the procedure. An adapter which can be relied upon to prevent such annoying incidents is presented. This apparatus presents the following characteristics:

(1) It is simple in construction.
(2) It is standardized for use with the ordinary equipment found in any hospital.
(3) It fits any surface.
(4) It is easily and readily applied.
(5) It allows no movement of the needle.

The instrument consists of two parts (Fig. 1). (*a*) The body which contains a straight central bore of caliber sufficient to accommodate the largest needle; (*b*) a flexible base carrying two lateral projections or arms. This base can be easily molded to conform to any surface. It is held in place by a rubber band which engages each of two inwardly facing hooks and which encircles the forearm.

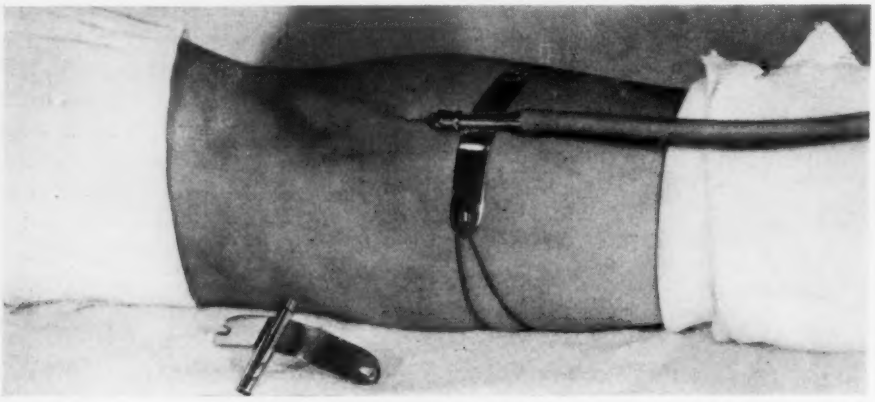

FIG. 1.—Showing adapter fitted to arm.

One end of the body fits any needle of standard size; the other end will admit an adapter or syringe. Annular heels are also provided to insure air-tight connection with rubber tube. The body is so constructed and applied to the base that the angle between the body and the base is such as to keep the end of the needle in the lumen of the vein and not allow it to come in contact with its wall.

Not only can this adapter be used with any type of transfusion apparatus, but it is equally available for use in connection with intravenous therapy of any sort.

AN IMPROVED CLAMP FOR THE MIKULICZ PROCEDURE

FRANCIS M. FINDLAY, M.D.

SANTA BARBARA, CALIF.

THE Mikulicz method of extraperitoneal resection of the colon has resulted in decreased mortality from peritonitis and shock. In this procedure the portion of the bowel to be removed is brought outside of the abdomen and the general peritoneal cavity closed about the colon. Often the lumen of the bowel is not opened for 48 hours and peritonitis from fecal contamination should not occur. This multiple stage operation has extended this field of surgery to include the poorer risk patients, who previously would not have been subjected to an extensive one stage operation. These weaker patients can now safely undergo resection in stages.

Surgeons have been deterred from wider use of the Mikulicz procedure because of difficulty in reestablishing the continuity of the bowel. Most patients dislike even a temporary colostomy. When delay and difficulty are experienced in closure of the colostomy, both patient and surgeon are dis-

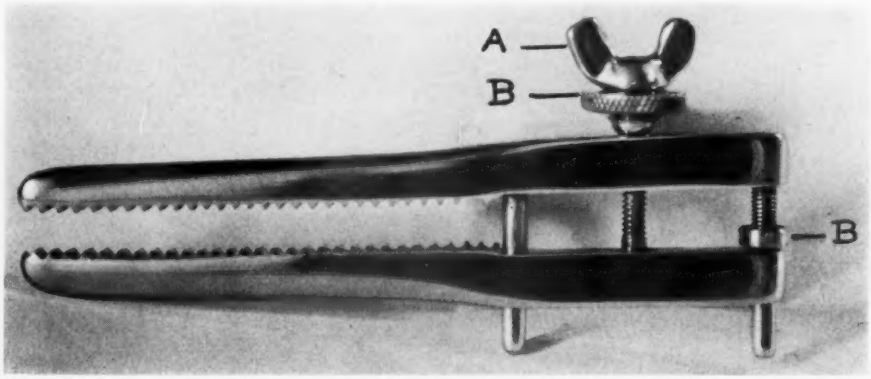

FIG. 1.—(A) Adjusting nut to keep jaws parallel and give equal pressure along jaws. (B) Lock nuts.

appointed and there is often a tendency on the part of the surgeon to do a one stage operation with greater risks from peritonitis.

We believe that much of the present dissatisfaction with the Mikulicz operation is from failure of the present methods to remove enough of the partition between the two limbs of bowel that form the colostomy. At the initial operation, two limbs of the colon on either side of the bowel to be removed are sutured parallel to each other for a distance of several inches. After the diseased portion of bowel has been removed, the septum formed by suturing these two limbs of colon must be removed. Enough of this bowel must be destroyed or the fecal stream will continue to appear at the abdominal stoma instead of following the normal route down the colon to the rectum.

We believe that the short narrow clamps now in general use have failed to remove enough bowel and are responsible for much of the present dis-

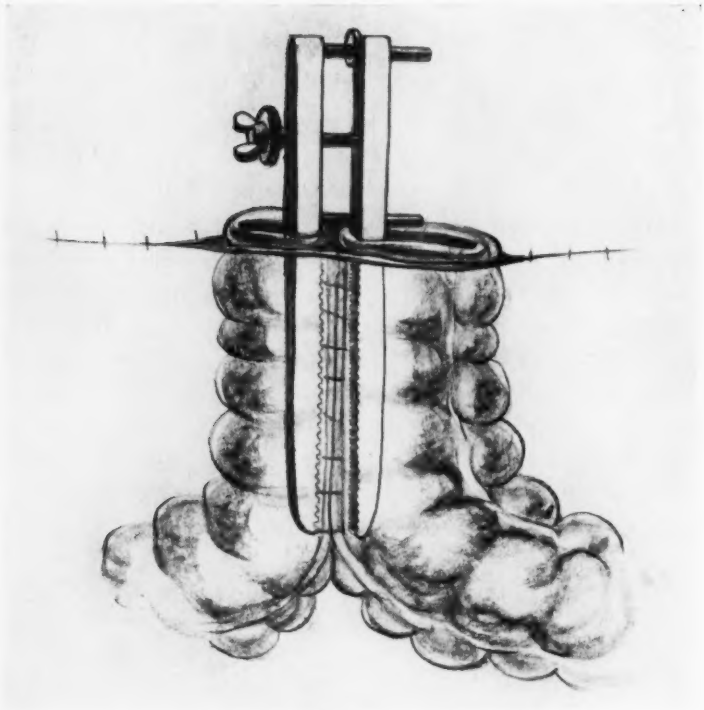

FIG. 2.—Mikulicz clamp in position.

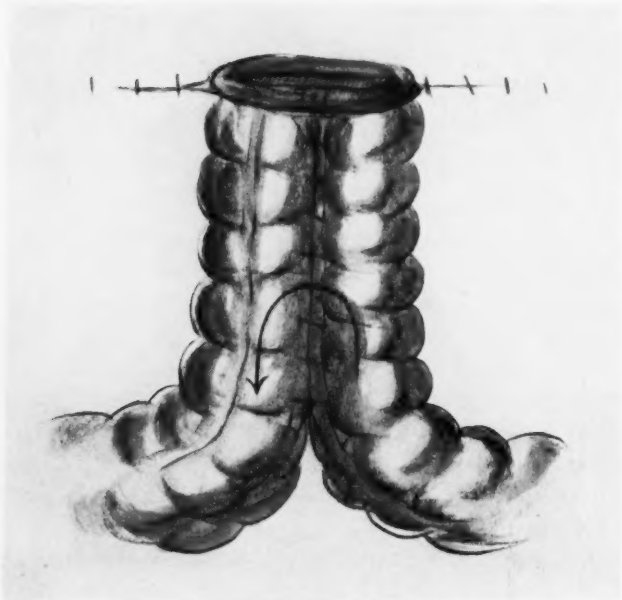

FIG. 3.—Clamp has come away. Showing resultant reestablishment of bowel continuity.

satisfaction with this type of operation. The clamp described below was devised to remedy this difficulty, and we believe it is superior to the Kocher or kidney clamps now in general use. It is essential that the clamp have both long and broad crushing surfaces to destroy enough bowel by pressure necrosis. This clamp is five and one-half inches long with crushing surfaces three and one-half inches long and one-quarter of an inch broad and will destroy enough bowel, even in obese patients with thick abdominal walls, to restore the fecal stream to its normal channel. The jaws have a spread of one inch and the two blades come apart so that they may be inserted separately and then locked in position if this is desired. The teeth are deep enough to securely engage the bowel walls and when the clamp is applied the bowel will not slip. An adjusting nut provides a means of maintaining equal pressure along the crushing surfaces, regardless of the thickness of the bowel (Fig. 1).

This short clamp does not protrude a great distance above the abdominal wall, is not cumbersome, and is easily covered by an abdominal pad. This is a great convenience when the clamp is applied to ambulatory patients. Once applied, no further attention is necessary except to keep the lock nuts tight so that the septum of bowel will be necrosed (Fig. 2). This usually occurs from the fifth to seventh day when the clamp comes away voluntarily, leaving a wide and deep channel for the fecal stream (Fig. 3). If closure of the colostomy is then desired, it may be done immediately by one of the accepted methods now in general use.

I am indebted to Mr. Sam Crist of the Engineering Department of the Cottage Hospital for his aid and technical suggestions in constructing this clamp.

MASSIVE HEMORRHAGE FROM POSTERIOR DUODENAL ULCER*

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CASE REPORT.—J. P., male, 58 years of age. Thirty years ago he complained of symptoms characteristic of ulcer; *i.e.*, discomfort about two hours after meals, relieved by food or alkalies, belching of gas and sour material, with periods of remission and freedom from symptoms. On October 22, 1919, he was admitted to the Lankenau Hospital on the service of Dr. John B. Deaver. For three to four years previously he had noticed that his stools were black at times and he would occasionally vomit coffee ground material. Three days before admission he vomited some blood and fainted. Recovering consciousness he had a similar attack three hours later, vomiting a teacupful of bright blood, and again fainted.

On admission he was obviously exsanguinated. His physical examination was otherwise negative. A diagnosis of bleeding gastric or duodenal ulcer was made and he was placed on a medical regimen under which he improved rapidly.

Eleven days later, Doctor Deaver operated upon him. The stomach, duodenum,

* Read before the Joint Meeting of the Philadelphia Academy of Surgery and the Boston Surgical Society, February 4, 1935.

gallbladder, liver and spleen all appeared normal. The stomach was opened and explored but no lesion found. The appendix, which showed a chronic condition, was removed.

Eleven months later he was readmitted with recurrence of gastric symptoms and black blood in his stools. Roentgenologic examination showed some constriction of the duodenum, thought to be due to adhesions. The stomach emptied completely in four and one-half hours. Bearing in mind the previous negative exploration, a tentative diagnosis of purpura hemorrhagica intestinalis was made and medical treatment instituted. His symptoms disappeared, the bleeding stopped, and the follow up five months later found him well.

His history from that time on may be abbreviated as one of intermissions with complete absence of symptoms interrupted at intervals of 6 to 12 months by periods of gastric symptoms with melena and gastric hemorrhage.

When first seen by the author in October, 1933, he had been well for many months, until two weeks before admission to the Abington Memorial Hospital, when his stomach symptoms had returned in severe form, followed two days before admission by vomiting of coffee ground material and on the day of admission by a severe gastric hemorrhage of a quart and one-half of bright, red blood. His hemoglobin was 62 per cent; red blood cells, 2,930,000; white blood cells, 21,600; coagulation and bleeding time, normal. Blood continued to be present in his stool and ten days later his hemoglobin had fallen to 52 per cent, but the red blood cells were still 2,920,000 and the white blood cells had fallen to 7,000. His general condition seemed much improved. Roentgenologic examination showed complete emptying of the stomach in six hours and no persistent defect of the stomach or duodenum. It was thought that he had a chronic posterior ulcer of the duodenum. Eight months later his gastric symptoms returned and he had a severe hemorrhage by mouth and bowel for which he was readmitted to the Abington Hospital in a condition of marked exsaguination. His hemoglobin was 34 per cent; red blood cells, 2,140,000; white blood cells, 19,500. He was given fluids parenterally and a transfusion the following day.

On June 8, 1934, his abdomen was reopened through the former scar and dense general adhesions encountered, involving the pyloric portion of the stomach and the duodenum. These organs with the transverse colon and the gallbladder were bound together and to the inferior surface of the liver. There was no evidence of cirrhosis. The spleen was not enlarged. There was no diverticulum of the small intestine. The gallbladder was dissected free from enveloping adhesions. It was not inflamed and contained no stones but it was thought best to remove it. No lesion was found in the stomach. The duodenum was then freed and no sign of disease found on the anterior wall nor could a crater be detected on the posterior wall. At a point just about opposite the right border of the gastrohepatic omentum, an area of inflammatory infiltrate was found and it was decided to open the duodenum to inspect its interior at this point. This revealed a large ulcer, almost one inch in diameter, on the posterolateral wall. In its bed could be dimly seen a vessel passing from above downwards. The ulcer bed was partly in the pancreas and extended upwards and to the right where the duodenal wall was guarded by the attached gastrohepatic omentum. It had eroded through the entire thickness of the duodenum and it was thought to be in intimate relation to the common duct. Safe excision seemed out of the question. As we have learned that gastroenterostomy frequently fails to relieve such a situation, it was decided to destroy the vessel with the actual cautery, to lightly cauterize the remaining ulcer bed and to perform a wide pyloroplasty to check the bleeding and to create conditions that would favor healing under an ulcer regimen (Fig. 1). He made an uncomplicated recovery and has been symptom free since.

Follow Up.—February 12, 1936. The patient is now symptom free and has had no further bleeding, the longest period without hemorrhage, since his original operation 16 years ago.

It is too soon to say that this procedure will be permanently effective.

He has had other long remissions. He illustrates, however, some of one's difficulties and perplexities. Just what to do with this type of ulcer, fortunately rare, is not a subject for dogmatism. I seriously doubt if these low chronic posterior ulcers heal under medical management. When they bleed recurrently as this one did they certainly become surgical. Possibly gastro-enterostomy by its change of chemistry and partial diversion of gastric contents, combined with medical measures, will sometimes bring about healing

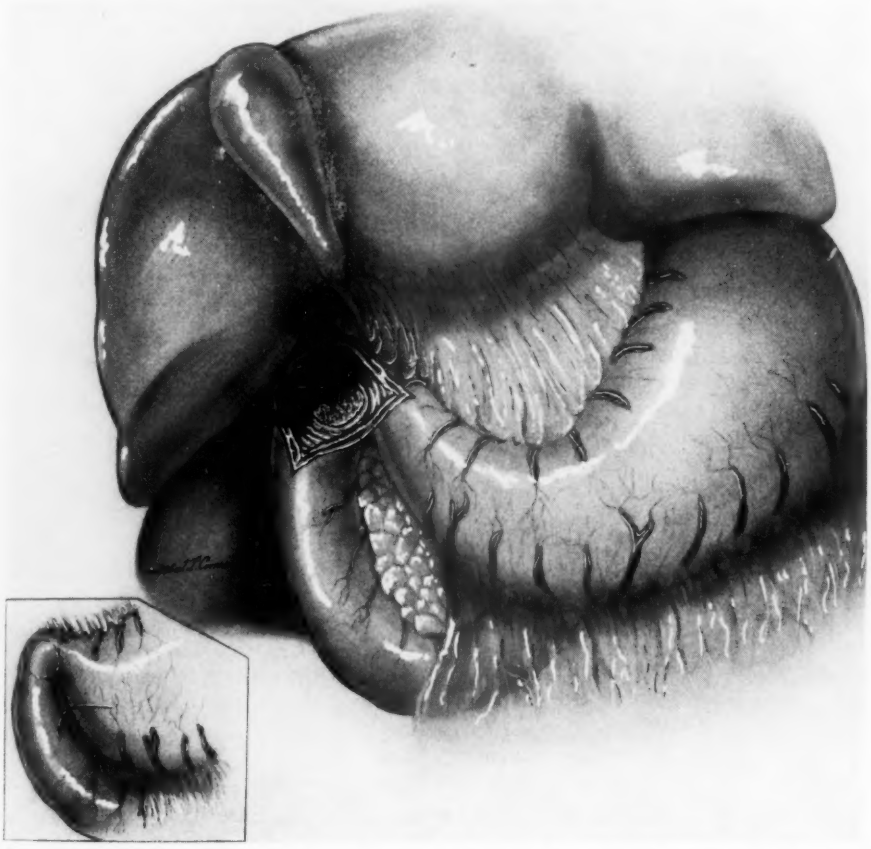


FIG. 1.—Approximate position and size of posterolateral ulcer. Inset shows large gastroduodenostomy with arrow suggesting probable diversion of gastric contents away from ulcer bed.

of the smaller and less indurated lesions but it is not reliable and introduces the danger of gastro-enterostomy complications. When the ulcer is fairly high and small it may be possible and preferable to excise the involved portion of the duodenum, but in the cases that I have seen such an attempt is fraught with great danger. Simply to open the duodenum and ligate or cauterize the bleeding vessel may check the hemorrhage but would seem to offer little towards cure. Several years ago I thought that the Devine procedure would short circuit the ulcer and permit healing under permanently alkaline conditions in the duodenum, but after encountering jejunal ulcer following this operation I became skeptical of its value. It

now seems to me that it is worthwhile to attack these ulcers directly through the opened duodenum. The smaller ulcers may at times be excised and partly closed. The larger ones may be cauterized and their margins nicked as chronic ulcers have been treated from time immemorial. Naturally caution must be employed in this cauterization. Then a large pyloroplasty is made both because such a closure is safer than a simple suture of the duodenum and because thereby a free interchange of gastric and duodenal contents is established, and thus alters the chemistry of the stomach juices and also a certain diversion of the course of gastric contents is accomplished which may favor the natural healing efforts of the tissues. I now have a few cases of this kind under observation in whom the results up to date seem highly satisfactory. Medical management following operation is of course obligatory.

It is my opinion that this plan will result in cure in a considerable number of these difficult cases without undue surgical risk and that the more resistant cases may be excised more safely after this preliminary attack, if it be found necessary.

Discussion.—Dr. David Cheever (Boston, Mass.).—It seems to me that this problem is so difficult and unsettled that I predict that 20 years hence the discussion of peptic ulcer will be equally interesting because it seems to me that it can never be standardized. We all look with envy on the statistics from some clinics which report 85 to 90 per cent of five-year cures of peptic ulcer either gastric or duodenal by simple gastrojejunostomy. We all have immediate satisfactory results after these operations but subsequently many of us have such a high percentage of marginal and duodenal ulcers at or near the stoma that we have become discouraged. There is therefore a widespread conviction that peptic ulcer should be submitted to a long medical regimen before being subjected to surgery. Doctor Pfeiffer's most interesting case brings up one of the greatest dilemmas which we face: What to do when confronted by massive hemorrhage from the upper alimentary tract? There are a number of very interesting things brought out by the story of his patient which might be emphasized. One is that massive gastric hemorrhage, if not due to ulcer, could probably be readily explained by simple exploratory operation. Therefore if we operate in such a case and do not at once find the explanation, we are certainly justified in making a very extensive exploration of the stomach and duodenum as Doctor Pfeiffer did. In this connection it is well to be reminded of the possibility of hemorrhage from the gastro-intestinal tract due to the ulceration of ectopic gastric mucosa in a Meckel's diverticulum. Doctor Allen, of Boston, has studied and contributed largely in an important way to the treatment of massive hemorrhage from peptic ulcer and his decision, as I understand it, is that in these cases the thing to do is not to wait too long in the hope that hemorrhage will cease spontaneously but to transfuse the patient, open the stomach and duodenum freely, in order to find the bleeding point. Then to resect the pylorus and first part of the duodenum and restore continuity, either by direct anastomosis or by gastrojejunostomy. I do not believe that it is ever possible permanently to stop this hemorrhage by local treatment of the ulcer after opening the viscus—that is, by cauterizing the base of the ulcer or by direct ligation. This simply will not succeed because after temporary arrest the gastric juice will digest away the ligature and the clot and bleeding will start up again. Another thing, we must not be too em-

phatic in our teaching that most cases of massive hemorrhage from peptic ulcer will recover if submitted promptly to medical treatment. Within a week or two I have seen a bleeding duodenal ulcer treated medically without avail and then subjected to transfusion with benefit but with final secondary hemorrhage and death. Formidable though the prospect is we ought early to feel the obligation to explore cases of persistent or massive hemorrhage presumably from ulcer. Drastic operation is justified in that type of case. My preference, in spite of Doctor Pfeiffer's experience with marginal ulcer, would be to divide the pylorus, resect it if reasonably feasible but at any rate to turn the ends in and restore continuity by gastrojejunostomy. It is my opinion that by diverting the acid gastric contents from the ulcer it will heal without the necessity of actual excision, though it is doubtless desirable.

Dr. E. L. Young, Jr. (Boston, Mass).—We see cases done by all of the different surgeons at all periods and with all types of surgery and I think we have come to formulate the conditions as follows: Duodenal ulcer as such is a medical and not a surgical disease. The complications are surgical only because we are either dealing with an unknown disease, of which the ulcers are the outward manifestation, or else we are dealing with a disease of multiple etiology, in which we cannot know the etiology in every case and can only treat the manifestation. We feel that surgery is indicated only in four conditions: (1) perforation; (2) pyloric obstruction; (3) hemorrhage; (4) that case disabled by pain or where symptoms of the ulcer are such that the patient cannot continue his or her occupation.

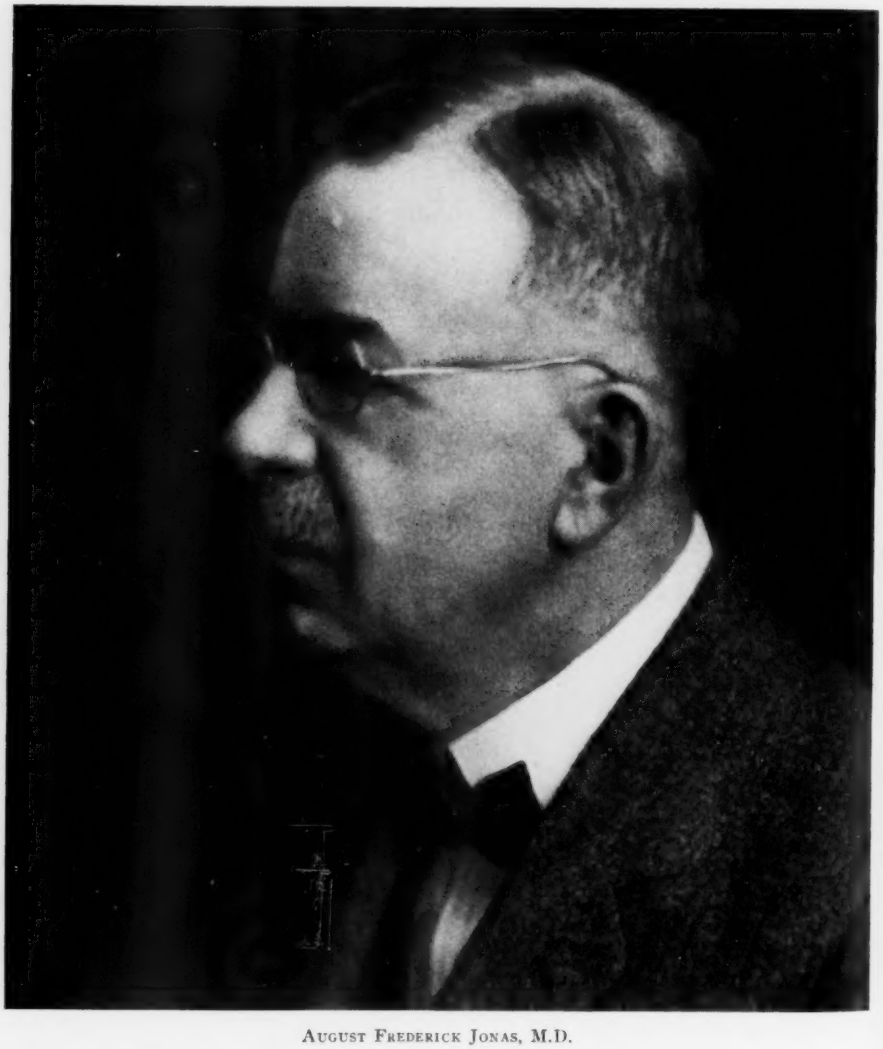
Regarding the hemorrhage, I think there are two or three things which are of importance. First, the age of the patient and second, the severity of the hemorrhage. Dr. Arthur W. Allen, in reviewing these cases at the Massachusetts General Hospital recently, found that in patients under 50 the primary hemorrhage was rarely fatal. The older the patient the greater the danger. The more hemorrhages there have been, the more of a risk. Consequently, we feel that those patients who have had a massive hemorrhage and recovered without operation or those who show evidence of repeated bleeding should be offered radical surgery; *i.e.*, resection of the pylorus and the turning back of the duodenum, and the tying of the blood supply from the posterior side. He told me he has had up to date 31 such cases without a single death. Twice in the past year I have performed radical surgery, once being forced in on account of acute hemorrhage; and both of these cases showed only very small ulcers of the anterior wall.

MEMOIR

AUGUST FREDERICK JONAS

1858–1934

AUGUST FREDERICK JONAS was born at Arlington, Wisconsin, in 1858. He died from cerebral hemorrhage at his home in Omaha, Nebraska, on November 12, 1934. Seven years previously he had suffered from a small cerebral



AUGUST FREDERICK JONAS, M.D.

hemorrhage which left an aphasia which caused his retirement from active work.

He was graduated from the Bennett Medical College in Chicago in 1877 and took up general practice in Wisconsin. In 1882, he went abroad and studied in Vienna, Berlin and Paris for a short time and then entered the Ludwig Maximilian University in Munich as a student and was graduated in 1884.

Returning to his home state of Wisconsin he again carried on a general practice until 1887 when he moved to Omaha and became connected with the Union Pacific Railroad, eventually becoming Chief Surgeon of the road and holding this position for 29 years, until his retirement because of his ill health. He served also as division surgeon for the Chicago and Northwestern Railroad and as assistant surgeon for the Chicago, St. Paul, Minneapolis and Omaha Railroad.

Being interested in teaching, Doctor Jonas joined the faculty of the Omaha Medical College as Professor of Clinical Surgery in 1892. In 1899, he added to his duties as a teacher the responsibilities of the deanship of the college and remained in that position until 1902 when the college was merged with the Medical Department of the University of Nebraska. He remained as Professor of Surgery and Chairman of the Department until his retirement in 1929, when he was given the appointment of Professor Emeritus. In 1927, he received the degree of Doctor of Laws from the State University.

Doctor Jonas was a member of many medical societies and was a conscientious attendant at meetings. He contributed to the societies by reading papers, entering freely into discussions, and serving in an official capacity when called upon. He was elected a Fellow of the American Surgical Association in 1901 and served as vice-president in 1906. He was a member of the Société Internationale de Chirurgie. At one time he was vice-president of the American Medical Association. He served for five years on the Board of Governors of the American College of Surgeons. He was president of the Western Surgical Association in 1901 and at various times served as president of the Missouri Valley Medical Society, the Nebraska State Medical Society and the Omaha-Douglas County Medical Society.

Doctor Jonas was essentially a clinical surgeon. Coming to Omaha in the early years of the city he identified himself with the surgical progress of the Middle West and gave of himself unsparingly to the efforts of the community to found hospitals and establish a medical college which would be a credit to scientific medicine.

Doctor Jonas' contributions to medical programs were concise, simply stated and written in a way to be understood readily. As he was an accomplished linguist, speaking both French and German almost as well as he did English, his acquaintance with foreign surgical literature was considerable and he added to his understanding of foreign methods by frequent trips abroad and personal contact with leaders in surgery.

As a teacher Doctor Jonas was highly respected by his students. His command of words was such that he was an adept in explaining the points that he wished to emphasize in such a way as to make the subject easily under-

standable. Consequently his clinics and ward rounds were well attended and popular with his students.

During the World War Doctor Jonas was commissioned Captain in the Medical Reserve Corps. He was active in organizing an ambulance company connected with Base Hospital No. 49 and acted as medical adviser to the Governor of Nebraska on military matters.

Doctor Jonas was a great believer in travel as a means of education. He regularly took a vacation each year and traveled to many parts of the world. On these trips he gathered material which appealed to his many interests. His collections, though not large, were brought together with discrimination, and are of great interest. They include paintings, Chinese jades, objects from the tombs of old Egypt, Alaskan Indian material and a large collection of geologic specimens from many countries. The acquisition of these collections enlarged his interests and made him a man of broad culture well grounded in many fields outside of his own science.

It is given to but few men to leave as great an impress on the community in which they lived as did Doctor Jonas. He came to a young and growing part of our country and by constant striving and continued hard work became an integral part of it and did much toward raising the standard of surgery during its period of rapid growth.

Doctor Jonas is survived by his widow, two sons and a daughter. His eldest son, August Frederick Jonas, Jr., is at present one of the residents in the Department of Surgery at Johns Hopkins University.

ALFRED J. BROWN, M.D.
Kansas City.

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